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Synopses

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Postgraduate ANZSPD Essay Competition Prizewinner

Over the last decade, endodontic treatment has changed significantly with access to technologies including NiTi files, reciprocating motors and advanced obturation techniques and materials. Discuss the application of this advance to primary dentition endodontics, indications and contraindications and alternate treatment modalities.

Dr Gregory Celine
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Abstract

The field of endodontics has presented many advancements that have had a downstream influence on how primary tooth endodontics is performed. These advancements can be found both in regard to chemomechanical preparation, such as in the case of rotary NiTi instrumentation, reciprocating motors and modern irrigants; as well as in obturation, in the form of new obturation materials and techniques that are applicable in the unique task of placing obturation materials into the torturous canals of primary teeth. Advancements to which access has improved over the last decade are presented herein and discussed in light of the associated evidence.

Introduction

Advancements in endodontics are almost universally found in the field of permanent tooth endodontics in adults. While there are indeed advancements particular to primary tooth endodontics, many advancements in this field occur downstream from mainstream endodontics. This report will deal with advancements in primary tooth endodontics, regardless of their origin. To address the issue of access in the last decade, materials or techniques included herein will not be limited to those whose first reports are found in the last decade, but will include those for which there is a body of supporting literature found in this time frame. Advancements related to chemomechanical preparation and those relating to obturation will be discussed separately.

Rationale for primary tooth endodontics

Early loss of primary teeth is most commonly due to dental caries or a traumatic dental injury 1. In many cases, loss of teeth is avoidable with the application of endodontic techniques, pending restorability of teeth in each case. Early tooth loss can result in many sequelae, including effects on aesthetics, speech2, loss of arch length[3,4], loss of arch circumference[5,6], particularly in younger patients and in cases where the successor is not close to eruption[7,8], and midline deviations[9].

For these reasons, it is in the interest of treating dentists to try, where possible, to maintain primary teeth in their position in the dental arch until natural exfoliation can occur.. A particular scenario where this may be especially important, is in cases where no successor exists. Excluding third molars, worldwide prevalence of permanent tooth agenesis has been reported at 2.5-6.9%, depending on the population10. An interdisciplinary approach to treatment planning cases of permanent tooth agenesis is recommended, usually in consultation with an orthodontist, as there are many considerations that should be made. Long-term retention of the primary tooth is sometimes not possible and at other times,

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THIS ISSUE

by Dr Sue Cartwright

BDS, Dip Clin Dent, M Ed

As 2018 draws to close we take a moment to reflect on the year.

In 2018 Colgate supported 6 Alliance for a Cavity Free Future Grant projects and the selection process for projects for 2019 has been completed. We will soon announce the successful applicants. If you wish to apply for a grant please email susan_cartwright@colpal.com and submit an application by the 1 Nov 2019 for the 2020 grants.

This year Bright Smiles Bright Futures reached 1 million kids around Australia and New Zealand with messages about good oral health. Dr Rabbit was very busy visiting schools and dental events!

We hope you had the opportunity to undertake a community project this year, with donated Colgate brushes and paste to assist implementation, either for Dental Health Week in August or for World Cavity Free Future Day on the 14th Oct. This year the World Cavity Free Future campaign reached 4 million people encouraging them to drink water instead of sugary drinks.

We hope 2018 was a good year for you all. We wish you the very best for the festive season and look forward to seeing you at the RK Hall Lecture Series in WA in 2019.

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not the best approach11, 12. Endodontic treatment approaches to retained primary teeth will be discussed later in this report.

Challenges in primary dentition endodontics

A sound understanding of the root canal morphology and variations that exist is paramount for clinicians undertaking endodontics in both permanent and primary teeth. While literature on the root canal morphology of permanent teeth has historically been plentiful, primary teeth have not traditionally been paid the same attention. Recent studies employing the use of three-dimensional (3D) imaging have definitively demonstrated the variation that exists in the root and root canal morphology of primary molars, both in number and shape.

In a study using cone beam computed tomography (CBCT) studies taken of patients being treated for other problems, Ozcan et al13 found that the presence of a "round" root canal was the least common canal shape. More common were ovoid or flat-ovoid canals. This was true for almost all canals of all primary molars from either arch. They also found that regardless of canal shape in the cervical and middle regions of the canal, the apical region almost always had an ovoid shape.

In maxillary molars, fusion of the distobuccal and palatal roots has been reported from around 17%13 to 27%[14] with communication variably reported between the canals of each.

Having an appreciation of the variability in primary molar root canal anatomy is important, particularly with the benefit of 3D imaging techniques, as very few situations exist in which clinicians have access to 3D imaging prior to embarking on endodontic treatment of primary molars. Usually, clinicians rely on two-dimensional (2D) imaging, so it is important to understand the possible anatomical variations in light of the shortfalls of 2D imaging.

Other challenges thought to be associated with pulpectomy of primary teeth are the inherent physiological root resorption and the close proximity of the crown of the successor tooth[15].

New technologies in endodontic chemomechanical preparation

Apex locators

Oznurhan et al16 reported a recent in vivo study where they compared electronic apex locators (EAL) to radiographic assessment of working length (WL) as a gold standard and found no significant difference between modalities. A shortcoming of radiographic assessment of WL that is acknowledged by most authors is the issue of root resorption of primary teeth. In both pathological and physiological root resorption, clastic activity resorbs dentine in a non-linear and inconsistent fashion[17]. In many cases, the apical foramen of the root canal can be found significantly further up the length of the root than the radiographic apex, increasing the chance of extending instruments and extruding irrigants and materials past the apex[18]. Coll and Sadrian19 reported a significantly lower success rate for pulpectomies in cases of teeth with more than 1mm of root resorption. Bodur et al18 reported an ex vivo study of two EAL's compared to visual assessment of the file exiting the apex in extracted teeth. They found varying success with the two EAL units used, however they found that there was no significant difference in success in roots with resorption compared to those without. Significantly, their sample included both anterior and posterior teeth – not the case for most studies.

Rotary NiTi instruments

First introduced to paediatric dentistry in a case report by Barr in 200020, rotary root canal instrumentation with nickel-titanium (NiTi) files is thought to be of particular benefit in primary tooth endodontics. By its ability to exist reversibly in two atomic conformations, namely austinsite and martinsite, NiTi has two advantageous properties over traditional stainless steel (SS) files, i.e. super-elasticity and shape memory. These properties make it ideal for use in endodontics with a rotary handpiece as it is better able to withstand cyclical loading and fatigue much more slowly than SS21.

The body of literature on the cleaning effectiveness and the relative time efficiency of rotary instrumentation (an important consideration when treating children), compared to hand instrumentation, have been investigated extensively. Reports can be found that conclude in favour of both NiTi rotary technology[22] and hand-filing with SS files[23], which highlights the lack of consistency in study protocol. One issue that has been discussed[24] is the role of individual investigator's experience and expertise using the newer technologies. Another is the inherent variation that exists between the different rotary NiTi file systems. For this reason, many studies compare more than one file system.

Despite methodological inconsistencies, there is a growing body of in vitro and in vivo evidence in support of rotary NiTi instrumentation during primary tooth pulpectomy. One such in vitro study is that by Musale and Mujawar[22] who compared hand instrumentation with K-files to rotary instrumentation with ProFile, ProTaper and Hero Shaper file systems. They found that the rotary NiTi systems universally demonstrated more conical canal preparation form with improved cleaning efficacy compared to hand instrumentation with SS K-files. Kuo et al[25] conducted an uncontrolled in vivo trial using ProTaper files with a modified protocol for use in primary molars. They used a two-step approach with 1:5 Buckley's formocresol as an inter-appointment dressing and Vitapex calcium hydroxide-iodoform paste for obturation and reported 96% clinical and radiographic success at 12 months. Pinheiro et al[26] assessed removal of Enterococcus faecalis (E faecalis) using scanning electron microscopy (SEM) and found that their hybrid NiTi-SS protocol was most effective, but NiTi alone was still more effective and quicker than SS hand-files alone. In particular, they found that hand-files left a significantly thicker smear layer containing bacteria and bacterial toxins. They also acknowledged that the increased removal of E faecalis in the hybrid system may be due to the increased number of filing steps with irrigant used between each step.

There are currently no rotary NiTi file systems marketed specifically for use in primary teeth. While a number of authors have described protocols for use with rotary file systems, some indeed being hybrid protocols employing the use of both SS hand-files and NiTi rotary files, much confusion and variation still exists for practitioners seeking a consistent protocol. As the field of rotary endodontics in primary teeth grows, it is possible that a system may eventually be designed and marketed specifically for use with primary teeth.

Reciprocating motors

One of the goals of chemomechanical preparation of root canals is maintenance of the overall canal path, to avoid canal transportation or strip or zip perforations. Although more literature exists in the realm of permanent tooth endodontics, in vitro studies have been published that assess the cleaning efficacy and the maintenance of the canal path for reciprocating NiTi systems compared to "conventional" rotary systems. Pinheiro et al27 compared efficacy of E faecalis

removal with ProTaper (a conventional rotary NiTi system) and WaveOne (a single-file NiTi reciprocating file system), and found no significant different in E faecalis removal, despite significantly reduced working time for the single-file reciprocating system. Prabhakar et al28 conducted a CBCT analysis of centring ability of two single-file NiTi systems – WaveOne, a reciprocating file system, and One Shape a rotary file system. They found that the reciprocating file system was significantly better at maintaining the canal form with less canal transportation than the rotary system. More high-quality research is needed in this area, initial in vitro reports of reciprocating file systems show promise for this newer method of employing NiTi files.

Ultrasonic instrumentation

The use of ultrasonic K-files has also been reported by some authors. Canoglu et al[29] found in their in vitro study that use of ultrasonic with SS K-files demonstrated significantly more zip perforations and shortening of working length than rotary NiTi and conventional hand filing with SS K-files. Da Costa et al[30] conducted an uncontrolled trial of 18 pulpectomies in primary molars using SS K-files with ultrasonic activation, followed by obturation with a calcium hydroxide-iodoform paste, and they reported only one failure after 14 months of follow-up. More high quality literature would be needed to support use of this treatment modality. Canoglu et al's report indicates that ultrasonic instrumentation with SS K-files may have an inherent weakness that would be difficult to overcome given the inherent properties of the materials involved.

Irrigants

Irrigants have multiple purposes in endodontics, such as instrument lubrication, antibacterial activity, and dissolution of organic or inorganic material. Dissolution of inorganic material has been the focus of recent work surround the removal of the smear layer in the root canal system of primary and permanent teeth in endodontics. A systematic review and meta-analysis by Shahravan et al[31] found that the evidence supports an improved fluid-tight seal of the root canal system of permanent teeth. In primary teeth, it has been demonstrated that 6% citric acid presented efficacy in smear layer removal in primary tooth root canal dentine[32]. However, in vivo trials have shown that smear layer removal has not affected the success of ZOE pulpectomies[33,34].

In most protocols, the root canal irrigant of choice is sodium hypochlorite (NaOCl), with some protocols also supporting the use of normal saline[15]. Due to the thin root dentine, there is believed to be an increased emphasis on canal irrigation in chemomechanical preparation of primary teeth, compared to permanent teeth. A newer irrigant to make its way from permanent tooth endodontics is MTAD (a mixture of a tetracycline isomer, citric acid and a detergent). One report using qPCR to compare efficacy against E faecalis of 2.5% NaOCl and MTAD found them to be equally effective[35]. Another report used only 1% NaOCl and found that it was not as effective at reducing the overall intracanal bacterial count as MTAD[36].

New technologies (materials and techniques) in obturation

Historically, the most common pulpectomy obturation medicament has been zinc-oxide eugenol (ZOE). Other agents have been added to the ZOE, such as formocresol as described by Coll et al[37], however these have largely faced other challenges that are not in the scope of this paper. In recent times, more modern materials and techniques have been presented in the literature that may provide benefits over ZOE.

Materials

Due to its historical standing as the staple pulpectomy material, ZOE is still the yardstick against which most newer materials are compared in the literature.

One of these newer materials is combination calcium hydroxide/iodoform paste. Randomised controlled clinical trials have demonstrated increased success of these pastes compared to ZOE. One of these trials was that by Trairatvorakul and Chunlasikaiwan[38] who studied 54 mandibular primary molars that were treated with pulpectomy using conventional SS K-files, followed by obturation with either ZOE or Vitapex (a calcium hydroxide/iodoform paste). The combined clinical and radiographic success of ZOE at six and 12 months was 48% and 85%, respectively; compared to 78% and 89%, respectively, for Vitapex. The better success of Vitapex in this study was in agreement with the results of Mortazavi and Mesbahi[39], who reported 78.5% overall clinical and radiographic success for ZOE and 100% for Vitapex after 10-16 months of follow-up. Interestingly, Mortazavi and Mesbahi also noted that in the case of Vitapex, any material that was noted to be extruded in immediate post-operative radiography was found to be completely resorbed, often as early as three months post-operatively. This was not the case for ZOE where very little resorption of extruded material was noted, with some cases showing particles of ZOE still present after exfoliation of the crown of the tooth and eruption of the permanent successor.

A broad group of materials that has made its way to primary tooth endodontics from permanent tooth endodontics is antibiotic pastes. Numerous formulations are available, with scope for even more, given the broad range of antibiotics available that can be compounded into pastes relatively easily. In a clinical trial, Nakornchai et al[40] reported no statistically significant differences between the clinical and radiographic success rates of 3Mix (a triple antibiotic paste of metronidazole, minocycline and ciprofloxacin) and Vitapex; although it should be noted that the radiographic success rate in this study was not as good as in the studies described above[38,39].

Aminabadi et al41 published the results of their trial comparing the use of 3Mixtatin (the 3Mix combination described above, combined with simvastatin) and mineral trioxide aggregate (MTA) in treatment of primary molars showing radiographic signs of inflammatory root resorption. The 80 teeth in this trial are ones that would normally be considered hopeless and indicated for extraction, many with inflammatory root resorption severe enough as to cause perforations in the root dentine. At 24 months, 96.8% of 3Mixtatin teeth showed clinical and radiographic healing, whereas no teeth in the MTA showed radiographic healing with almost half of the teeth showing clinical signs of pain, mobility of sinus tracts.

An alternative method for pulpectomy that uses these antibiotic pastes is lesion sterilisation, tissue repair (LSTR), Takushige et al[42] described the use of this technique in an uncontrolled clinical trial wherein 3Mix was mixed as an ointment or with root canal sealer and placed into "medication cavities" of 1mm diameter, 2mm depth (where possible) or simply over the canal orifices in the pulp chamber. Out of 87 molars treated, all eventually achieved clinical resolution of symptoms, but the protocol was inconsistent in terms of how many visits were needed, and four

teeth required re-treatment as clinical resolution of symptoms did not occur after the first treatment.

Obturation techniques

Most obturation materials used in primary tooth endodontics are in the form of a paste. Traditionally, a lentulo spiral, either hand-operated or motor-driven, has been used for placement of the obturating material into the canal. Recently, other techniques have been suggested and trialled to aid with improving the quality of fill of the canals, such as different types of syringes and other motor-driven devices similar to the lentulo spiral.

Several recent in vitro studies exist that employ the use of 3D radiographic techniques to assess quality of fill. Nagaveni et al[43] and Singh et al[44] used spiral computed tomography and CBCT, respectively, and both found that the lentulo spiral provided the most optimal fill with least voids, although they disagreed on whether hand-operated or motor-driven lentulo spirals were superior. Grover et al[45] conducted an in vivo study of anterior and posterior teeth and assessed the quality of fill with 2D radiography and found that the least number of voids and most canals with optimal fill were achieved with Pastinject, a motor-driven device that functions similar to a lentulo spiral.

Retained primary molars in cases of agenesis of the succedaneous premolar are a challenge for paediatric dentists. A study by Bjerklin and Bennet[46] assessed the long-term survival of mandibular second primary molars in such a predicament. They found that if the primary molar is still present when the patient is 20 years old, then the long-term prognosis is good. In cases of retained primary molars that require endodontic treatment, obturation with gutta percha, in a style similar to endodontic treatment of permanent teeth has been recommended[47]. In a slightly novel approach, O'Sullivan and Hartwell[48] obturated a retained primary molar with MTA. They chose the material based on its sealing properties but did not address its shortcomings such as technique sensitivity and high cost.

Conclusion

Many advancement in endodontics have made their way into primary tooth endodontics, downstream from permanent tooth endodontics. Advancements in both the chemomechanical preparation and obturation stages of endodontic treatment can be seen emerging in the literature. The last decade has seen access to many of these advancement improve as they become more mainstream. In many areas, further research is needed to solidify the body of evidence surrounding use of these advancements in primary teeth.

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ANZSPD President's Report

Dr Sue Taji

Sitting in an airport lounge in Bangkok after having just attended the first Global Summit on Early Childhood Caries, one cannot help but appreciate that there are many like-minded clinicians and researchers in all corners of the globe working in unison, with one vision, the betterment of the oral health of their paediatric patients.

Following IAPD's invitation to national and regional society presidents from 69 member societies to attend the meeting with IAPD Board members and other national society presidents, 'the IAPD Global Summit was held in the first week of November in Thailand. The Congress brought together international experts from far and wide and member societies all put forward and discussed current concepts and approaches within their regions.

It was intriguing to hear of the many regions of the world in which poverty still first and foremost is the inhibiting element to access oral health care. Irrespective of the level of income, sugar is however still considered to be the main driving force in Early Childhood Caries, with alarming consumption levels per capita evident across the world. In Australia, based on the most recent national data regarding per capita sugar consumption, Australians have 60 g of sugar per day and alarmingly this raises to 92 g per day in teenage males (14-18 year olds). Lack of knowledge amongst parents, the wider community and governments at all levels have likewise contributed to difficulties in reducing rates of Early Childhood Caries.

As clinicians, our focus is often on the local level, what we provide our patients and their parents and the community we live in, and as such it was refreshing to hear first hand from other member nation presidents what was occurring in other regions on a global level. As was discussed at the congress in Thailand, the prevention of Early Childhood Caries should be everyone's glocal business, where 'Glocal' is the global ambition delivered in a locally appropriate way. ANZSPD is a path for all of its members to be involved and contribute to what happens on a regional level across Australia and New Zealand.

Recently ANZSPD accepted an invitation to collaborate with the Academy of Child and Adolescent Health (ACAH), an academy that was launched in Australia in late 2016 for all those working in healthcare with newborns, children and young people. The ACAH includes medical, dental, allied health and other associations to promote the health and wellbeing of every newborn, child and young person. Such collaborations will in future increase the strength of our voice and our visions in promoting the oral health of children, particularly when advocating policy or other strategic directions with government bodies and key decision makers.

With the year rapidly drawing to a close, preparations are well underway for the RK Hall event in Perth and I hope to see all of you at the RK Hall event in a couple of months time, in mid-March. The event will bring together an elite group of experts and will provide a wonderful learning experience for all attending.

Wishing you all a safe and happy Festive Season and a good start to the new year,

Kind regards,

Dr Sue S. Taji
ANZSPD President

NZ Branch Report

Tēnā koutou,

The ANZSPD New Zealand Branch has had a more relaxed year in 2018 after a busy year in 2017 with hosting the RK Hall lecture series in Auckland.

The NZ branch has been active on the research front and is sponsoring a summer studentship position at the Hutt Hospital. We have a wonderful 4th year dental student helping us with a research project on the dental late effects of childhood cancer. We look forward to hearing the results of this study in 2019.

Our annual study day, is always a highlight on our calendar, and this year has been no exception. This is a wonderful day where our members' come together and listen to some organized speakers but can also present cases or topics if they choose in a very supportive environment. I would like to acknowledge our wonderful speakers in Wellington this year, they showed the depth of talent we have in our organisation.

Our AGM held after the Study Day in Wellington this year was a very special one. Four of our hard working and loyal members were made Honorary Life Members of the NZ Branch to honour and thank them for all their service to the NZ Branch ANZSPD and Paediatric dentistry. Congratulations to Drs Alan Isaac, Mary Anne Costelloe, Erin Mahoney, and Craig Waterhouse.

So it is with a heavy but also lighter heart I end my 2 year tenure as president. Mike Brosnan I wish you well in this role. Wishing everyone all the best for the festive season.

Ngā mihi,
Kate Naysmith

ANZSPD Essay Competition Prizewinner

Bioactive and biomimetic dental materials have advanced from relatively specialised highly biocompatible low strength medicaments to include newly emerging restorative materials. Explain the definition of bioactive and biomimetic in the context of these materials and consider how the well developed and newer materials may be utilised in paediatric restorative dentistry

Jamie Leung
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Abstract

As we move away from GV Black's "extension for prevention", there is now increasing emphasis on preservation of tooth structure to maintain the biomechanical, functional and aesthetic integrity of the tooth. Initially the ideal dental restorative material was considered one that was biologically inert and therefore biocompatible. But now there appears to be an evolving trend away from more inert compositions to those capable of interacting directly with tooth tissue, to form a bond between the tooth and the material (bioactive materials). Further to the developments in this newly emerging category of dental materials, there also appears to be a need for materials that mimic more closely the structure and function of natural enamel and dentine (biomimetic materials). Evidently, in paediatric dentistry the two most significant reasons for clinical failure of direct restorations is secondary caries and fracture of the restoration[1,2], which among other factors, may both be indirectly attributed to the mismatch between the biomechanical properties of the restoration and tooth tissue. For this reason, in addition to endodontic applications such as pulp capping and pulpotomy medicaments, the newly emerging bioactive and biomimetic dental materials may play a role in improving restoration survival and the longevity of restored primary and permanent teeth. Overall, significant in vitro data exist regarding the biological and biomechanical properties of these materials, but more clinical data is required to validate use of these bioactive and biomimetic materials in restorative applications.

Introduction

The concept of restorative dentistry has shifted away from GV Blacks "extension for prevention". Now, there is increasing emphasis on preservation of tooth structure to maintain the biomechanical, functional and aesthetic integrity of the tooth. Together with increased emphasis placed on minimal intervention in the management of carious and non-carious tooth substance loss, continuing advancement of dental materials has made preservation of sound tooth structure and maintenance of pulp vitality possible. Initially the ideal dental restorative material was considered one that was biologically inert and therefore biocompatible. But now there appears to be an evolving trend away from more inert compositions to those capable of interacting directly with tooth tissue (bioactive materials). Further to the developments in this newly emerging category of dental materials, there also appears to be a need for materials that mimic more closely the structure and function of natural enamel and dentine (biomimetic materials). Most of the biological and mechanical reasons for clinical failure of direct restorations can be attributed to the inability of the restoration to function like and be harmonious with the natural tooth structure. Evidently, in paediatric dentistry the two most significant reasons for clinical failure of direct restorations is secondary caries and fracture of the restoration[1,2], which among other factors, may both be indirectly attributed to the mismatch between the biomechanical properties of the restoration and tooth tissue. For this reason, the newly emerging bioactive and biomimetic dental materials may play a role in improving restoration survival and the longevity of restored primary and permanent teeth. This can have a significant impact on the wellbeing of the child in the long term, as masticatory ability, phonetics, aesthetics and space maintenance can be maintained in the growing child.

This essay will discuss the definition of bioactive and biomimetic in the context of dental materials, and will review the literature of the more well developed materials for use in paediatric restorative dentistry.

What are bioactive and biomimetic dental materials?

The terms 'bioactive' and 'biomimetic' each have their own definition in the context of dental materials. But it is often difficult to separate these dental materials exclusively based on the individual definitions, as bioactive dental materials can also have biomimetic properties and vice versa. Although the definitions are defined below, it is important to note that there is usually overlap in the properties found in these newly emerging dental materials.

Bioactive materials are not simply those that interact with the underlying tooth structures e.g. fluoride release, adhesion to tooth structure. A bioactive material was defined by L Hench (1971) as a material that "elicits a specific biological response at the interface of the material which results in the formation of a bond between the tissues and the material".[3] A bioactive material should be capable of stimulating specific biological responses through biochemical and biophysical reactions that result in the formation of an apatite layer.[4] This allows for integration of the biomaterial into the environment, which in this case would be the dental hard tissues. However it must be noted that despite this capability, the host response to the biomaterial i.e. the extent of hydroxyapatite formation is dependent on the innate and nonspecific immune responses occurring in the surrounding tissues.[4] Bioactive materials are divided into osteoconductive

materials, where the surface is colonised by osteogenic stem cells, and osteoproductive materials, where bone migrates along the biocompatible surface.[4]

On the other hand, biomimicry is the study of the structure and function of natural biological designs so that it can be imitated and improved in synthetic compositions. In restorative dentistry, the unquestionable reference is the intact natural tooth. The definition of biomimetic dental materials can therefore be used to represent either the manner of material processing similar to the natural process within the oral cavity, or a material that "mimics" or recovers the biomechanics of the original tooth tissue. With this requires the understanding of both the composition, arrangement and physical properties of dental hard tissue so that it can be imitated. The ideal biomimetic restorative material aims to restore the structural and physical interrelation between an extremely hard tissue (enamel) and a more resilient softer tissue (dentine), together with the composition and properties of each tissue independently and its relationship to the remaining tooth structures.

Is there a need for these materials in restorative dentistry?

With continued improvement in adhesive procedures and further development of restorative materials, the aesthetic and biomechanical behaviour of the enamel-dentine complex can be partially recovered. However despite this, restorative materials are still lacking – firstly, in their ability to completely recover the biological and mechanical properties of the original tooth structure, and secondly, in forming a sound bond to the underlying tooth. The aforementioned limitations can manifest clinically as secondary caries and fractures of the restoration, the two most significant reasons for clinical failure of direct restorations. In paediatric restorative dentistry, 52% of compomer restorations in primary posterior teeth and 52% of composite restorations in permanent posterior teeth were replaced due to secondary caries when observed over a period of five years.[2] Similarly, the most common reason for failure of conventional and resin-modified GICs was secondary caries followed by bulk fracture of the restoration.[1] This leads us to question the integrity of the 'bond' that these adhesive restorations are understood to provide. This is in line with that seen in the permanent dentition, where secondary caries was found to be the most common reason for failure in direct posterior composites when observed over a period of seven years. In fact, histological studies of composite resin adhesion to tooth structure show that complete replacement of the demineralised etched surface with resin is practically unattainable.[6]

For this reason, though continual improvement in these conventional restorative materials may address these issues, there is potential for developmental of newer restorative materials with different compositions and mechanisms to 1) provide a more secure interface between the restoration and tooth structure, and 2) improve the biological and mechanical properties of the restorative material so that it is more compatible with original tooth structure. One hypothesis is that bioactive materials would make it more difficult for secondary caries to form because the formation of hydroxyapatite between the restoration and tooth should create a more secure interface. Through this, secondary effects may arise such as improvement in dentine hypersensitivity, bond strength and the potential for repair/regeneration of dental hard tissue at the restoration-tooth interface. The latter is important in paediatric dentistry for pulp preservation. It facilitates continued root development and apexogenesis in immature teeth. All of which are critical for long term retention of a permanent tooth.

Bioactive and biomimetic dental materials

Bioactive materials are predominantly considered in endodontic therapy due to their low compressive strength and long setting times. But newly emerging bioactive materials show improved strength and physical properties that may be adopted for use in restorative dentistry. The literature on the following classes of bioactive and biomimetic materials and their uses in paediatric restorative dentistry will be explored: calcium hydroxide, calcium-silicate based cements, calcium-aluminate based cements, bioactive glasses and crystalline calcium phosphate materials.

Calcium hydroxide

Calcium hydroxide has been used in dentistry for many decades and is still being widely used for its bioactive properties. Aside from use as a medicament, root filler and for repair of perforation in endodontic therapy, calcium hydroxide is commonly used as a restorative liner in deep cavities to stimulate mineralisation of the subjacent dentine. It is also used following carious and traumatic pulp exposures for pulp preservation. Dentine bridge formation can be attributed to the dissociation of the calcium and hydroxyl ions, which decrease the concentrations of inhibitory pyrophosphates to cause mineralisation.[7] Calcium hydroxide has also been shown to maintain odontoblast vitality where the remaining dentine thickness is less than 0.5mm.[8] When no pulp exposure was present, calcium hydroxide exhibited the greatest reactionary dentine deposition when compared to RMGIC, ZOE and zinc polycarboxylate.[9] The dissociated calcium ions are capable of stimulating differentiation of stem cells from the pulp and induction of dentine-pulp repair.10 Calcium hydroxide can therefore be considered for use in paediatric restorative dentistry as 1) protective liner in deep cavities without pulp exposure, 2) indirect pulp capping agent when there are no signs of pulp involvement, 2) direct pulp capping agent following mechanical or traumatic exposure and 4) pulpotomy medicament, in both primary and young permanent teeth. At this point in time, the use of calcium hydroxide as a direct restorative material has not been consistently validated due to its low elastic modulus and compressive strength, its high water solubility and inability to bond to dentine or resin-based restorative materials.[11] For this reason, when used as a liner it is critical to cover the calcium hydroxide with a restorative material that is capable of providing a seal to prevent microleakage. Calcium hydroxide also exhibits antimicrobial properties[12], which suggests potential for decreasing the extent of bacterial induced pulpal inflammation and reducing the incidence of secondary caries beneath direct restorations.

Calcium-silicate based cements

Calcium silicate materials are derived from Portland cement, the basic building material. They are heterogeneous in composition and contain varying proportions of calcium oxide/silicon dioxide and calcium aluminate.[13] Mineral trioxide aggregrate (MTA) has a predominant silicate component and was the first calcium silicate material to develop into a viable material for clinical use. The original gray MTA (GMTA) and more aesthetic white MTA (WMTA) have both shown bioactive properties that make it suitable for use following mechanical and traumatic pulp exposure. When GMTA and tooth structure were exposed to phosphate-buffered physiological solutions in vitro, there was formation of a white material at the interface between the GMTA and dentine.[14] Bozeman et al showed that under the same conditions, crystal precipitates of both WMTA and GMTA

were chemically and structurally similar to hydroxyapatite.[15] This leads us to consider that further developments in MTA may allow for a restorative material that integrates into the tooth tissue to enhance its adhesion. When compared to calcium hydroxide, dentine bridge formation by MTA was more homogenous and less porous.[16] In paediatric restorative dentistry, MTA is used as a direct pulp capping agent and pulpotomy medicament for pulp preservation and formation of a reparative dentine bridge. When used in pulpotomy of primary and permanent teeth following carious and traumatic exposures, MTA has shown higher long term success when compared to calcium hydroxide.[17] With MTA there is greater predictability in dentine bridging and pulp health. But tooth discolouration has been reported when used for revascularisation.[18] As with calcium hydroxide, at least 1.5mm of MTA should cover the exposure and surrounding dentine and covered with RMGIC. It should be noted however that at this point in time, uses of this material beyond pulp capping have not been consistently validated due to the slow initial setting time of 3-4 hours and low compressive strength of 20-60mPa.[13]
Biodentine is another calcium silicate cement with a wider range of clinical application. In addition to the established clinical uses of MTA, Biodentine can be used in paediatric restorative dentistry to restore large coronal, cervical and radicular lesions.[13] It has improved biological and biomechanical properties when compared to the other calcium silicate cements. It's faster setting time of ~12 minutes, development of early strength and higher reactivity can be attributed to the addition of a predominant tricalcium silicate component.[13] With regards to the restorative indications, Biodentine has been advocated for use as a permanent dentine replacement material. It is not suitable for use as a permanent enamel replacement material as it is less stable than composite resin.[19] For this reason Biodentine can be used as a temporary posterior restoration in stress-bearing areas but should later be cut back to serve as a base beneath the composite resin. When evaluated clinically, Biodentine showed very good marginal adaptation, surface finish and no post-operative sensitivity when used in Class I and II posterior restorations.[20] However this clinical evaluation involved a small sample size (19) and a short follow up period of 6 months. Its suitability as a posterior restorative material can be validated by its marginal integrity comparable to that of RMGIC, colour stability and improved compressive strength.[21,22] The elastic modulus, microhardness, flexural and compressive strength of Biodentine is comparable to that of natural dentine.[19] In the presence of simulated body fluid Biodentine was also capable of stimulating deposition of hydroxyapatite on the cement surface.[23] Indicating that in addition to use as a direct pulp capping agent, there is potential for integration of the restorative material into the underlying tooth tissue, increased bond strength and perhaps less sequelae from poor marginal sealing.
Bioaggregrate is another calcium silicate cement which can be used as a pulp capping agent in paediatric restorative dentistry. With regards to composition, Bioaggregrate is different to MTA in that there is no calcium aluminate. It also includes added calcium phosphate constituents. When used as a pulp capping agent, Bioaggregrate shows greater potential in induction of odontoblastic differentiation and mineralisation when compared to MTA.[24] This ability to stimulate hard tissue formation at the interface of the restoration and tooth tissue can help maintain pulp vitality following mechanical exposure. Though Bioaggregrate has shown good biocompatibility, potent antibacterial action and excellent sealing ability in vitro, it's application as a restorative material is limited by its low compressive strength.[4]

Calcium-aluminate based cements

These materials appeared approximately 8-10 years after the introduction of calcium-silicate cements. Like calcium-silicate cements, calcium-aluminate cements contain calcium oxide/silicon dioxide and calcium aluminate. The difference is that they have a higher proportion of aluminate compared to the other constituents. Doxadent (DD) is a calcium-aluminate based cement intended for use as a direct restorative material. It has been advocated for use in the restoration of Class I, II and V cavity preparations.[25] In vitro studies of an experimental calcium aluminate dental restorative material show compressive and flexural strengths greater than a RMGIC but less than a nano-hybrid composite.[26] Hardness was reported to be superior when compared to the same materials.[26] However resistance to wear was relatively low.[26] Contrary to these improved mechanical properties observed in vitro, clinical studies may avert use in stress-bearing areas. When DD was used as a posterior restoration over three years, there was a cumulative failure frequency of 72.6%.[27] Main reasons for failure included material or tooth fracture, particularly in Class II cavities. Therefore uses may be limited to non-stress bearing indications in paediatric restorative dentistry.

Calcium phosphate materials

Calcium phosphate materials are said to be both bioactive and biomimetic. Hydroxyapatite is the most frequently used calcium phosphate material due to its excellent biocompatibility and its ability to be osteoconductive. Formation of a reparative dentine bridge in vitro was quicker and thicker with tricalcium phosphate-hydroxyapatite when compared to calcium hydroxide.[28] This suggests potential for a more effective and efficient direct pulp capping agent. Hydroxyapatite may also be used as a filler in composite resin to improve mechanical properties and remineralisation potential.[29] With regards to composition, synthetic hydroxyapatite is similar chemically and structurally to natural enamel and dentine. Its biomimetic properties may also be attributed to lattice parameters similar to that of natural hard dental tissue.[29] However as with many of the other bioactive and biomimetic materials discussed, synthetic hydroxyapatite possesses low mechanical strength and fracture toughness which limits its use in load-bearing areas.

Bioactive glasses

Bioactive glasses show osteoconductive and osteoinductive properties. Following dissolution, a layer of hydroxycarbonate apatite forms on its surface. This is thought to be due to the stimulation of osteogenic bone cells to form bone matrix by the dissolution products of the glass.[30] The hydroxycarbonate apatite is structurally and chemically similar to the mineral phase of bone and is thought to integrate to the host bone through interaction with the collagen fibrils.[30] Since bone and dental hard tissue have similar chemical and structural compositions, there may also be a potential for integration of this material into the underlying tooth structure. However enhancement of its low mechanical strength and fracture toughness would extend its scope of application in restorative dentistry.

Conclusion

Although significant in vitro data exist regarding the biological and biomechanical properties of these materials, more clinical data is required to validate use of these bioactive and biomimetic materials in restorative applications. The formation of an apatite layer at the restoration-tooth

interface and mimicking of the biological and biomechanical properties of hard tooth structure minimises the mismatch between the restorative material and the underlying tooth structure. With further improvements, it may be possible to use these bioactive and biomimetic materials in paediatric dentistry to 1) manage dentine hypersensitivity, 2) restore carious and non-carious tooth substance loss, 3) maintain pulp vitality following carious/mechanical/traumatic pulp exposure, 4) improve the marginal seal of direct restorations and fissure sealants and 5) minimise biological and mechanical failure of restorations resulting from mismatch with the underlying tooth structure during function.

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Developmental Defects of Primary teeth and Molar Incisor Hypomineralisation

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1. Introduction

Initiation of deciduous tooth development occurs in utero with enamel formation commencing at approximately 13-16 weeks; permanent tooth development commences at birth (Table 1) (Logan and Kronfeld 1933). Abnormalities in development can result in developmental defects of enamel which are not self-correcting and remain a permanent record on the enamel surface. The prevalence of developmental defects of enamel in Australian school children has been reported at 25% in the primary dentition and 58% in the permanent dentition (Seow, Ford et al. 2011).

2. Developmental Defects of Enamel

Amelogenesis occurs during the late bell stage of tooth development when ameloblasts differentiate from the inner enamel epithelium (Nanci 2017). Enamel formation is a two-step process with initially 30% mineralisation and then subsequent maturation and further mineralisation. Developmental defects of enamel (DDE) may occur from genetic changes or environmental (both local and systemic) conditions (Clarkson and O'mullane 1989). The presentation and severity of the defect is dependent on the stage of development at the time of insult and the extent and duration of the change.

There is a wide range of developmental defects of enamel with various aetiological causes. Genetic diseases can also disrupt key processes during tooth formation to result in characteristic phenotypes. Disruption early in initiation of the tooth bud can lead to agenesis or supernumerary teeth, disruptions during crown formation can lead to dentinogenesis imperfecta, dentine dysplasia or amelogenesis imperfecta. Defects of root formation may cause taurodontism or cementum agenesis. The World Dental Federation established the DDE index initially in

Deciduous Dentition	Enamel formation (in utero)	Crown Complete	Eruption
Central incisor	13-16 weeks	1-3 months	6-9 months
Lateral incisor	14-16 weeks	2-3 months	7-10 months
Canine	15-18 weeks	9 months	16-20 months
First molar	14.5-17 weeks	6 months	12-16 months
Second Molar	16-23.5 weeks	10-12 months	23-30 months
Permanent Dentition			
Mandible:			
Central incisor	3-4 months	4-5 years	6-7 years
Lateral incisor	3-4 months	4-5 years	7-8 years
Canine	4-5 months	6-7 years	9-11 years
First premolar	1.75-2 years	5-6 years	10-12 years
Second premolar	2.25-2.5 years	6-7 years	11-12 years
First molar	Birth	2.5-3 years	6-7 years
Second molar	2.5-3 years	7-8 years	11-13 years
Maxilla:			
Central incisor	3-4 months	4-5 years	7-8 years
Lateral incisor	11 months	4-5 years	7-8 years
Canine	4-5 months	6-7 years	11-12 years
First premolar	1.25-1.75 years	5-6 years	10-11 years
Second premolar	2-2.5 years	6-7 years	10-12 years
First molar	Birth	2.5-3 years	6-7 years
Second molar	2.5-3 years	7-8 years	12-13 years

Table 1. *Deciduous and permanent dentition tooth development with enamel formation, crown complete and eruption times. Adapted from (Logan and Kronfeld 1933)*

1982 which has been modified (Table 2) for epidemiological studies to provide some consistency through the literature (Weerheijm and Mejàre 2003). However, this index system does not differentiate between the broad range of aetiology for these defects and relies on clinical description. Under this index, the extent of the lesions are subdivided into less than 1/3 involvement (mild), at least one third to two thirds (moderate) and at least two thirds (severe) categories (Crombie, Manton et al. 2009).

Modified DDE index	Code
Normal	0
Demarcated opacities:	
White/cream	1
Yellow/brown	2
Diffuse opacities:	
Diffuse – Lines	3
Diffuse – Patchy	4
Diffuse – Confluent	5
Confluent/patchy + staining + loss of enamel	6
Hypoplasia:	
Pits	7
Missing Enamel	8
Any other defects	9
Extent of Defect	
Normal	0
<1/3	1
At least 1/3 <2/3	2
At least 2/3	3

Table 2. *Modified Developmental Defects of Enamel Index (Weerheijm and Mejàre 2003).*

2.2 Clinical Presentations of DDE

The presentation and severity of DDE are dependent on the stage of enamel development at the time of the insult as well as the extent and duration of the insult. Ameloblasts secrete proteins such as amelogenin and ameloblastin to form an enamel matrix and regulate the removal of water and proteins from the enamel matrix with promotion of mineral ingress; the ameloblasts are sensitive to changes in their environment, including minor physiological changes which may result in enamel defects seen histologically or clinically (Nanci 2017). These defects can be broadly categorised into qualitative and quantitative defects (Seow 2017). Qualitative defects are usually associated with altered enamel mineralisation and may present as changes in translucency or opacity of the enamel which may be well demarcated or diffuse and coloured white, cream, yellow or brown. It is thought that qualitative defects are more likely to occur from disturbances in the final stages of enamel formation. Quantitative defects usually arise from disruption of enamel matrix formation and may present as hypoplasia: pits, grooves and thin or missing enamel; this occurs at the earlier stage of enamel secretion. Amelogenesis requires a stable physiological environment and disturbances early or late in the formation are likely to cause different clinical manifestations.

The location of a defect may suggest the timing of the disturbance on enamel formation. The neonatal line is a band of abnormal enamel with disorganised prism alignment with a higher content of organic material and is present in almost all primary teeth (Sabel, Johansson et al. 2008). The neonatal line may be more pronounced if the child experienced adverse neonatal conditions such as foetal distress or difficult delivery (Seow 2017). Perinatal illness is often seen as an enamel defect at the neonatal line, continuing toward the root of the tooth. Diffuse opacities are thought to be related to a systemic insult with the group of teeth undergoing maturation at the same time, whilst demarcated opacities are more commonly noted in teeth with a localised or transient injury (Salanitri and Seow 2013).

2.3 Aetiology of DDE

The aetiology of DDE is still unclear, possibly due to the heterogeneity of index systems in population studies, or due to the vast range of hereditary, acquired, systemic and local factors which are associated (Salanitri and Seow 2013). Hereditary conditions may have enamel defects that involve only enamel or they may be a component of a generalised systemic syndrome. There are also many systemic or local acquired conditions that occur during the antenatal, perinatal or post-natal periods which result in DDE.

Hereditary conditions may be medical syndromes which feature DDE or be a dental only anomaly. Amelogenesis imperfecta occurs as a disruption to the genes which express for enamel formation. Defects may present as enamel hypoplasia, hypomineralisation or hypomaturation and characteristically are present for both primary and permanent dentitions. Medical syndromes which feature enamel hypoplasia are listed in Table 3 (Salanitri and Seow 2013).

Numerous acquired conditions from both systemic or local origin have been reported to cause DDE in the antenatal, perinatal and postnatal periods of development (Salanitri and Seow 2013). Prenatal conditions which have been associated with DDE include maternal vitamin D deficiency during pregnancy, maternal smoking during pregnancy, increased maternal weight gain during pregnancy, failure to access antenatal care and multiple births (due to the higher rate of neonatal complications) (Ford, Seow et al. 2009, Taji, Seow et al. 2011). Postnatal conditions linked to DDE are extensive and summarised in Table 4.

2.4 Prevalence of DDE

The prevalence of DDE in the primary dentition has not been well reported. (Salanitri and Seow 2013) Publications in the previous two decades have reported prevalence range between 10% to 49% globally, as summarised by Table 5. Australian studies are limited but DDE in the primary dentition has been reported at 25% for a cohort of 517 Queensland school children (Seow, Ford et al. 2011); demarcated opacities were predominant followed by diffuse opacities and missing enamel was most commonly enamel hypoplasia, followed by grooves and enamel pits.

Syndrome	Reported defects	Other Systemic Defects
Usher Syndrome	Enamel hypoplasia (de la Peña and Valea 2011)	Sensorineural hearing loss, retinitis pigmentosa
Seckel Syndrome	Enamel hypoplasia (Regen, Nelson et al. 2010)	Intellectual disability, multiple skeletal defects
Ellis Van Creveld Syndrome	Enamel hypoplasia (Nakatomi 2009)	Skeletal and cardiac system defects.Multiple frenula, congenital missing teeth, abnormal tooth morphogenesis
Treacher-Collins Syndrome	Enamel hypoplasia, enamel opacities (da Silva Dalben, Costa et al. 2006)	Tooth agenesis, ectopic eruption, isolated cleft palate, cleft lip and palate
Oto-dental Syndrome	Enamel hypoplasia (Colter and Sedano 2005)	Large bulbous crowns, deep enamel fissures, pulp chambers duplicated, supernumerary teeth
Velocardiofacial Syndrome (22q11 deletion)	DDE (Klingberg, Dietz et al. 2005)	Hypocalcaemia and hypoparathyroidism

Table 3. *Hereditary medical syndromes which feature enamel hypoplasia defects. Adapted from (Salanitri and Seow 2013).*

Acquired Condition	
Nutritional Deficiencies	Insufficient supply and absorption of vitamin A, C, D, calcium for preterm (Yengopal, Harneker et al. 2009) and indigenous children (Jamieson, Armfield et al. 2006)
Suboptimal nutrition	Prolonged breastfeeding without solid supplementation (Leviton, Rabinowitz et al. 1992)
Birth factors	Prematurity and low or very low birth weight (Seow and Wan 2000)
Abnormalities in mineralisation pathways	Hypocalcaemia, osteopaenia, rickets, hyperbilirubinaemia, renal and liver disease (Seow, Brown et al. 1984)
Insufficient absorption of minerals	Prematurity(Seow and Wan 2000), coeliac disease (Majorana, Bardellini et al. 2010)
Infectious diseases	Bacterial and viral infections of the urinary tract, otitis and upper respiratory disease (Ford, Seow et al. 2009). Congenital syphilis, chicken pox, rubella, measles, mumps, influenza, cytomegalovirus (Seow 1991).
Cerebral palsy	Caused by maternal or foetal infection, foetal anoxia, hyperbilirubinaemia (Bhat, Nelson et al. 1992)
Local trauma	Laryngoscopy and endotracheal intubation (Seow, Brown et al. 1984)
Chemical and drug exposure	Fluoride levels >1ppm (Wong, McGrath et al. 2006), lead levels from exposure to lead paint, accidental or pica-ingestion,(Seow 1991) tetracyclines, (Owen 1963) amoxicillin (difficult to discern from fever) (Hong, Levy et al. 2005)

Table 4. *Acquired conditions in the postnatal period of development associated with DDE. Adapted from (Salanitri and Seow 2013).*

Country	Year	Prevalence of DDE
Australia	2011 (Seow, Ford et al. 2011)	25%
Mexico	2011 (Casanova-Rosado, Medina-Solis et al. 2011)	10%
USA	2009 (Hong, Levy et al. 2009)	4%
Brazil	2007 (Chaves, Rosenblatt et al. 2007)	44%
USA	2003 (Montero, Douglass et al. 2003)	49%
USA	2001 (Slayton, Warren et al. 2001)	33%
China	1996 (Li, Navia et al. 1994)	22%

Table 5. *Prevalence of developmental defects of enamel in the primary dentition. Adapted from (Salanitri and Seow 2013).*

3. Molar-incisor-hypomineralisation

3.1 Terminology

Molar-incisor-hypomineralisation (MIH) is an established term to describe a range of developmental enamel defects on permanent first molars(Weerheijm, Jälevik et al. 2001). The defects have previously been described as "hypomineralised permanent first molars", "idiopathic enamel hypomineralisation in permanent first molars", "non-fluoride hypomineralisation in permanent first molars" and "cheese molars". The all-encompassing name, MIH, was proposed in 2001 for systemic origin of 1-4 permanent first molars, frequently associated with affected incisors to be used by clinicians and in epidemiological research.

The most recent European Academy of Pediatric Dentistry (EAPD) policy document on MIH acknowledges that demarcated opacities of the same type as in MIH have been observed on second primary molars, tips of permanent canine cusps, second permanent molars and premolars and therefore the name may require revision at some stage (Lygidakis, Wong et al. 2010). Although the term MIH is now established, there is a risk that the term is misleading and may result in an under-estimation of the defect.

3.1.1 Hypomineralised second primary molars

In the primary dentition, enamel hypomineralisation can be present on second primary molars (Elfrink, Schuller et al. 2008). The term hypomineralised second primary molars (HSPM) was put forward to recognise this phenomena as its own diagnosis, rather than classified through the broad DDE index (Weerheijm and Mejàre 2003). There are less studies into the aetiology of HSPM than MIH; The available studies are of good quality with low risk of bias and had positive associations of HSPM to similar aetiological factors as MIH (Elfrink, Veerkamp et al. 2006, Elfrink, Schuller et al. 2008).

Available evidence suggests second primary molars are greater affected by caries than first primary molars(Elfrink, Veerkamp et al. 2006); A positive correlation between enamel hypoplasia and caries in the primary dentition was found (Slayton, Warren et al. 2001) and there is an expected association between hypomineralisation in second primary molars and caries (Elfrink, Schuller et al. 2008).

A recent systematic review into the predictive relationship between HSPM and MIH found high heterogeneity between the 14 available studies (Garot, Denis et al. 2018). Despite the limitations of the studies and the difficulty in comparing number of participants and variable caries risk, the presence of HSPM was found to have a positive association with MIH prevalence.

3.1 Diagnosis of MIH and HSPM

The clinical features of MIH vary but are used to distinguish between other developmental enamel defects. Demarcated lesions of abnormal enamel translucency (opacity) contrast to diffuse opacities of fluorosis, chipping and exposure of dentine or unexpected caries. The breakdown of enamel can occur immediately after eruption or under masticatory forces which is distinguished from hypoplasia. Clinically, the teeth may be very sensitive to air, cold, heat and mechanical stimuli. Histologically, there are enamel porosities

Criteria	
0	No visible enamel defect
1	Enamel defect, not MIH/HSPM
11	Diffuse opacities
12	Hypoplasia
13	Amelogenesis imperfecta
14	Hypomineralisation defect (not MIH/HSPM)
2	Demarcated opacities
21	White or creamy demarcated opacities
22	Yellow or brown demarcated opacities
3	Post eruptive breakdown
4	Atypical restoration
5	Atypical caries
6	Missing due to MIH/HSPM
7	Cannot be scored

Table 3. *A proposed charting system to record observation as per the EAPD diagnostic criteria. Adapted from (Ghanim, Elfrink et al. 2015).*

of varying degree with normal enamel well demarcated; the carbon content of the enamel in the affected areas is higher than normal enamel and the calcium and phosphorus concentrations are lower (Jälevik, Klingberg et al. 2001). These clinical and histological features have been used to describe the phenomena of MIH.

The diagnostic criteria put forward by the EAPD in 2003 by a consensus meeting (Weerheijm and Mejàre 2003) was then modified in 2009 (Jälevik 2010) and 2015 (Ghanim, Elfrink et al. 2015). One to all four permanent first molars must show hypomineralisation of the enamel and simultaneously the permanent incisors may or may not be affected; the defect increases in severity when more molars and incisors are affected (Lygidakis, Wong et al. 2010). The charting criteria for examination is summarised in Table 3 and can be used for MIH or HSPM. The affected teeth show clearly demarcated opacities of various colour and size at the occlusal and buccal part of the crown. The colour may be white, cream, yellow or brown and may vary from negligible to majority of the clinical crown. Posteruptive breakdown, atypical restorations and extractions of permanent molars and incisors may occur. Severity can be classified as mild, when demarcated enamel opacities are present but without enamel breakdown, occasional sensitivity to external stimuli, or severe, when demarcated enamel opacities occur with breakdown, caries, persistent or spontaneous sensitivity affecting function.

As the number of permanent first molars affected with hypomineralisation increased, there was a subsequent increase in the involvement of the incisors (Oliver, Messer et al. 2014). The same Australian cohort found among first permanent molars affected, most of the defects were brown (47%), had cuspal involvement (74%) and included post-eruptive breakdown (67%). A Finish population of children found a higher prevalence of MIH in the maxilla when compared with the mandible (Leppaniemi, Lukinmaa et al. 2001). More recent research did not find a significant difference between maxilla and mandibular teeth or right and left side (Arrow 2008).

3.2 Aetiology of MIH

Molar-incisor-hypomineralisation is a distinct form of DDE in which the enamel of the first permanent molars with or without involvement of the central incisors is specifically hypomineralised (Weerheijm, Elfrink et al. 2015). Calcification of the first permanent molar commences from 3.5-4 months in utero until maturation completes 2.5-3years of age and these timings are very similar for the incisors (Logan and Kronfeld 1933). In vitro studies have suggested a narrower window of susceptibility at 6-8 months age based on histological, mechanical and chemical properties of affected first molar teeth (Fagrell, Salmon et al. 2013). The aetiology of MIH is likely to be caused by not one specific factor but several risk factors may act together to increase the risk of the individual (Crombie, Manton et al. 2009, Lygidakis, Wong et al. 2010).

The lack of standardised studies on MIH has led to low quality evidence on the aetiological factors associated (Elfrink, Ghanim et al. 2015). The suggested causes are similar to DDE in both primary and permanent dentitions and have been categorised by prenatal, perinatal and postnatal putative factors (Table 4) (Weerheijm, Elfrink et al. 2015). Earlier reviews subdivided the aetiological factors implicated in MIH into infant exposure to dioxins and biphenols, perinatal events, exposure to fluoride, childhood illness and specific chronic disease (Crombie, Manton et al. 2009).

A recently systematic review into the aetiology of MIH failed to find significant data on any aetiological factors (Silva, Scurrah et al. 2016). Prenatal factors which had been investigated were maternal smoking during pregnancy, maternal illness during pregnancy and maternal medication which all failed to find statistically significant results; maternal stress had higher odds of MIH but this has not been corroborated in any other studies (Ghanim, Manton et al. 2013). Perinatal exposures such as prematurity, low birthweight, caesarean delivery and birth complications were associated but with little evidence confirming significance (Silva, Scurrah et al. 2016). An older but significant study in the literature associated long duration of breastfeeding to MIH through the exposure of the infant to dioxins (Alaluusua, Lukinmaa et al. 1996). However, the same group discarded this association with a follow up study but attributed the non-significance in results to a reduction in levels of dioxin pollution (Laisi, Kiviranta et al. 2008). Early childhood illnesses were widely studied but ranged between specific conditions such as asthma and fever to studies which reported "general health" or "general illness" under age 3 to 4 years; childhood illnesses have a positive association without

Timing	Factor
Prenatal	Maternal illness or infection
	Maternal hypocalcemia
	Nutrition
Perinatal	Infant hypoxia
	Very low birth weight
	Premature birth
	Calcium shortage
	Medical problem (miscellaneous)
Postnatal	Breastfeeding
	Nutrition
	Calcium shortage
	Dioxins and polychlorinated bisphenols
	Environmental pollution
	Childhood illness (miscellaneous)
	Chicken pox and other viral infections
	Otitis media
	Asthma, lung problems, allergy
	Fevers (irrespective of cause)
	Medications (miscellaneous)
	Antibiotics
	Antiasthmatic medication

Table 4. *Commonly accepted factors associated with MIH. Adapted from (Weerheijm, Elfrink et al. 2015).*

significance to MIH (Silva, Scurrah et al. 2016). Genetic predisposition and epigenetic influences are likely to be part of the multifactorial origin of MIH but require further investigation. The lack of detail and consistency between exposure to environmental factors and recall of information in questionnaire format for majority of studies reduces the quality of evidence available for the aetiology of MIH.

3.3 Prevalence of MIH

The modified DDE index (Table 2) is often used for examination of MIH with demarcated lesions subdivided into mild, moderate and severe categories with severe including post-eruptive breakdown or existing restorations; a systematic review found that several studies excluded carious or restored teeth which is likely to lead to underestimation of the prevalence of MIH (Crombie, Manton et al. 2009). Severely compromised MIH teeth may be extracted at an early age, and this may not be accounted for using the modified DDE index.

The prevalence values for MIH vary widely (Jälevik 2010). Although the EAPD have provided criteria in which to score MIH for epidemiological studies, often studies have proceeded with the modified DDE index or studies with their own classification or sub-classification systems (Elfrink, Ghanim et al. 2015). Recommendations for prevalence study protocols have been suggested including a minimum of 300 children selected, age 8 years for optimum age of examination for MIH. With this basic criterium, previous prevalence studies have been rejected as well as the possibility of an increasing prevalence because this requires examination of the same cohort or population to report such a trend. Including EAPD and DDE index systems, overall prevalence ranged globally from 10-20% (Elfrink, Ghanim et al. 2015).

3.3.1 Global prevalence

Difficulty in reporting prevalence for populations come from the significant heterogeneity between participants and diagnostic criteria used in prevalence studies for MIH. Children in selected prevalence studies outlined in Table 5 find population studies range from 24 to 3 518 children. The lowest reported prevalence of MIH was in China and Hong Kong with 2 635 children were evaluated using the EAPD classification of MIH with a mean prevalence of 2.8% (Cho, Ki et al. 2008). The highest reported prevalence was from an Australian cohort of 24 children with a reported 44% prevalence using the modified DDE classification (Balmer, Laskey et al. 2005). Using the recommended minimum sample size of 300 children, the highest reported prevalence was 37.3% in Denmark using the EAPD classification (Wogelius, Haubek et al. 2008).

Country	MIH (%)	Criteria	Children	Reference
Australia	22	mDDE	550	(Arrow 2008)
Australia	44	mDDE	24	(Balmer, Laskey et al. 2005)
Bosnia	12.3	EAPD	560	(Muratbegovic, Markovic et al. 2007)
Bosnia	11.7	EAPD	104	(Mulic, Cehajic et al. 2017)
Brazil	40.2	EAPD	292	(Soviero, Haubek et al. 2009)
Brazil	19.8	EAPD	918	(da Costa-Silva, Jeremias et al. 2010)
Bulgaria	3.58	EAPD	2 960	(Kukleva, Petrova et al. 2008)
China	2.8	EAPD	2 635	(Cho, Ki et al. 2008)
Denmark	37.3	EAPD	745	(Wogelius, Haubek et al. 2008)
England	11	mDDE	3 233	(Balmer, Toumba et al. 2015)
Finland	17	Own	102	(Alaluusua, Lukinmaa et al. 1996)
Finland	19.3	Own	488	(Leppaniemi, Lukinmaa et al. 2001)
Germany	4.3-14.6	EAPD	2 395	(Petrou, Giraki et al. 2014)
Greece	10.2	EAPD	3 518	(Lygidakis, Dimou et al. 2008)
India	9.2	EAPD	1 366	(Parikh, Ganesh et al. 2012)
India	6.31	EAPD	1 792	(Mittal, Goyal et al. 2014)
Iran	20.2	EAPD	810	(Ghanim, Bagheri et al. 2014)
Iraq	21.5	EAPD	823	(Ghanim, Morgan et al. 2011)
Italy	13.7	Own	227	(Calderara, Gerthoux et al. 2005)
Jordan	17.6	EAPD	570	(Zawaideh, Al-Jundi et al. 2011)
Libia	2.9	Own	378	(Fteita, Ali et al. 2006)
Lithuania	9.7	EAPD	1 277	(Jasulaityte, Veerkamp et al. 2007)
Nepal	13.7	EAPD	749	(Shrestha, Upadhaya et al. 2014)
Netherlands	9.7	Own	497	(Weerheijm, Groen et al. 2001)
Netherlands	14.25	Own	442	(Jasulaityte, Weerheijm et al. 2008)
New Zealand	14.9	mDDE	850	(Mahoney and Morrison 2009)
New Zealand	18.8	mDDE	235	(Mahoney and Morrison 2011)
Spain	17.8	EAPD	550	(Martinez Gomez, Guinot Jimeno et al. 2012)
Spain	21.8	EAPD	840	(Garcia-Margarit, Catala-Pizarro et al. 2014)
Sweden	4.4 – 15.4	Own	2 252	(Koch, Hallonsten et al. 1987)
Sweden	18.4	mDDE	516	(Jalevik, Klingberg et al. 2001)
Turkey	14.9	EAPD	147	(Kusku, Caglar et al. 2008)

Table 5. *Overview of the global prevalence studies on MIH with the criteria and number of children in the study provided.*

3.3.2 Australian prevalence

Early Australian studies assessed the prevalence of DDE rather than the prevalence of MIH. A Melbourne population study found 82% of children with a medical comorbidity had DDE (Hall 1989). Another study of 517 Queensland school children found 25% prevalence in the primary dentition and 58% in the permanent dentition (Seow, Ford et al. 2011).

There are no available Australian prevalence studies investigating MIH using the EAPD criteria for assessment. A population study in Perth school dental service found a prevalence of demarcated opacities in permanent molars at 22% of the child population (using the modified DDE index system) and 47% of permanent molars noted to have diffuse enamel defects (Arrow 2008). A smaller Sydney cohort or 24 children used the modified DDE index system to reported 40% prevalence of MIH in children aged 8-16 years (Balmer, Laskey et al. 2005).

These children were a specific cohort undergoing orthodontic treatment and the prevalence rate is high in the published literature.

3.4 Alterations of MIH enamel

3.4.1 Structural Properties

Knowledge of the structural properties of the enamel of teeth affected with MIH is important in understanding the pathogenesis and to derive appropriate management. A recent systematic review assessed 23 studies on the structural, mechanical and chemical properties of MIH enamel (Elhennawy, Manton et al. 2017). The microstructure and mineral density of MIH enamel has been studied with a variety of methods such as light microscopy, polarised light microscopy, scanning electron microscopy and transmission electron microscopy (Elhennawy, Manton et al. 2017). Porosity was found to be increased in MIH enamel from 5-25% of normal enamel with creamy/white lesions and those without PEB being the least porous (Crombie, Manton et al. 2013). Enamel affected by MIH was found to have less dense prism structure, partial loss of prismatic pattern, loosely packed crystals and less distinct prism borders (Crombie, Manton et al. 2013, Bozal, Kaplan et al. 2015). The transitional area of clinically sound and healthy enamel adjacent to the demarcated MIH lesion was been found to have less mineralised prism sheaths than unaffected enamel even though it had a normal clinical appearance (Mahoney, Rohanizadeh et al. 2004). Porosity in enamel has been suggested to cause a subclinical inflammation in the pulpal cells of MIH teeth which can cause ongoing hypersensitivity and difficulty anaesthetising (Jalevik and Klingberg 2002).

3.4.2 Mineral Density

In vitro studies using x-ray micro-computed tomography have consistently found a decrease in mineral density in teeth affected with MIH with a mean of 19-20% (Elhennawy, Manton et al. 2017). Another study found average mineral content to be 59% vol mineral compared to normal enamel with mineral 86% (Crombie, Manton et al. 2013). The mineral density decreased from the CEJ to the occlusal surface and the density was highest near the DEJ (Farah, Swain et al. 2010). The sample sizes were low for the in vitro studies but provide a baseline understanding into the mineral properties of MIH teeth.

3.4.3 Mechanical Properties

The modulus of elasticity and hardness values for MIH enamel are significantly lower than those for normal enamel (Elhennawy, Manton et al. 2017). A number of investigations also found that the mechanical properties at the transitional region adjacent affected enamel also had significant reduction in mechanical properties when compared to sound enamel. The prismatic change in enamel of MIH is suggested to cause these changes to the mechanical properties; subsequent bonding can be ineffective for restorative management of these teeth (Jalevik and Klingberg 2002).

Ultrastructural changes occur in teeth with MIH, including in the areas of clinically sound enamel (Bozal, Kaplan et al. 2015). The etching pattern of hypomineralised enamel has been suggested to uniformly remove the enamel rather than the usual differential pattern of etched sound enamel. This is likely due to changes in ionic composition which affect the etching pattern and may interfere with adhesion to bonding materials. The surface of hypomineralised enamel contains increase proportions of carbon and oxygen and may indicate persistence of organic matter remains. This has been postulated as fault in the enamel maturation period during which the organic matrix is usually resorbed and mineral content of the hydroxyapatite (HA) crystals increases. The substitution or loss of carbonate in HA crystals is known to increase the solubility during acid etching. The classic type I and II etching patterns observed in the control tooth with normal enamel is thought to be what provides retention and clinical certainty of adhesion and marginal sealing.

3.5 Management Approaches

Management of MIH range from prevention, restoration to extraction and the decision is dependent upon the severity of the condition, the age of the patient and the expectations of the patient and parent (Lygidakis, Wong et al. 2010). Prevention is important at an early age to minimise the structural damage due to the porosity of MIH resulting in early caries or post-eruptive breakdown. As the child ages, prevention becomes less necessary and restorative management or extraction may be required.

It has been suggested that management can be divided dependent on severity of the MIH and into short-term and long-term treatment options (Mathu-Muju and Wright 2006). Mild MIH can be described as demarcated opacities in non-stress bearing areas of molars, isolated opacities, no history of sensitivity, no caries associated with defect and incisor involvement only mind if present; short-term treatment may be prevention, desensitising agents, fluoride varnish or sealants whilst long-term treatment has been suggested as ongoing preventative care. Moderate MIH involves intact atypical restorations, demarcated opacities present on occlusal or incisal third of teeth without PEB, PEB or caries limited to 1-2 surfaces without cuspal involvement, "normal" sensitivity and aesthetics of concern to the patient; short-term management may involve sealants and resin restorations for molars and bleach, fissure sealants, resin restorations and microabrasion for incisors whilst long-term management may involve ongoing prevention, full-cast coverage for molars or porcelain veneers for incisors. Severe MIH involves PEB, PEB on erupting tooth, history of sensitivity, widespread caries, crown destruction likely to advance to pulp, defective atypical restoration and aesthetic concerns; severe MIH may be managed short-term by GIC coverage, interim resin or stainless steel crowns for molars, bleaching and fissure seal, resin restoration or veneers for incisors and long-term management is likely to involve ongoing prevention, full-cast coverage molars or porcelain veneers or crowns for incisors.

3.5.1 Dental Anxiety

The change in morphology and ultrastructure of teeth with MIH produces issues with dentine hypersensitivity and difficulty bonding restorations which subsequently necessitates repeat restorative management with potential difficulty achieving analgesia (Alaluusua, Bäckman et al. 2001, Jalevik and Klingberg 2002). Behaviour management problems have been reported to be higher at 44% of a cohort of children with MIH compared to a control group at 2% (Jalevik and Klingberg 2002). This was suggested to be due to the repeated treatment of these teeth, often with accompanied pain during treatment. Interestingly, a 9 year longitudinal follow up of the same children found higher DMFT but no longer more dentally anxious than their non-MIH controls (Jälevik and Klingberg 2012). Early treatment planning and preventative strategies have been suggested as a way to reduced dental anxiety and behaviour management problems for these children.

Dental behaviour management problems in children with MIH has been associated with difficulty achieving analgesia (Jalevik

and Klingberg 2002). This has been attributed to subclinical inflammation of pulpal cells caused by the increased porosity of MIH enamel. Low age, parental fear, general anxiety, temperamental traits and painful dental treatments were all identified as risk factors for increased behavioural management problems and dental anxiety. Adjunctive management with nitrous oxide has been suggested as a potential to improve the effectiveness of local analgesia in children with MIH (Fayle 2003). The authors of this study found 51% of an Australian cohort of 283 children with MIH had treatment with nitrous oxide and local anaesthesia combination whilst 49% required treatment under general anaesthetic.

3.5.2 Prevention

Prevention may be advice to the parent or patient regarding dietary advice, fluoride toothpaste prescription, casein phosphopeptide-amorphus calcium phosphate (CPP-ACP) paste may help to remineralise and reduce caries experience on affected teeth (Lygidakis, Wong et al. 2010). The aim is not only to remineralise any demineralisation but also to address the mineral deficiency in the MIH lesion and adjacent enamel (Crombie, Cochrane et al. 2013). CPP-ACP with and without fluoride have been found to improve the mineral content and reduce porosity in MIH teeth.

Sensitivity in MIH teeth may be managed with professional prescription of fluoride. Porous enamel and post-eruptive breakdown can lead to pulpal inflammation and subsequent hypersensitivity or pain with chronic pulpal pain leading to local anaesthesia difficulties (Elhennawy and Schwendicke 2016). Early management for hypersensitivity is important in preventing poor oral hygiene, associated with sensitivity on brushing, and dental anxiety. A recent systematic review recommended remineralisation using CPP-ACP as a suitable modality to reduce mild to moderate hypersensitivity (Elhennawy and Schwendicke 2016). Spontaneous hypersensitivity may be managed by professional application of 22 600ppm fluoride varnish or 0.4% stannous fluoride gel (Lygidakis, Wong et al. 2010). Early preventative measures may allow the affected tooth to remain intact and promote maturation of the enamel so that maintenance of good oral hygiene alone is required and preventative steps reduced. Avoiding chronic pulpal pain is the main aim of addressing hypersensitivity early for these teeth.

3.4.3 Restoration

3.4.3.2 Surface Sealing

Teeth with mild MIH without caries or cavitation of enamel may be suitable for fissure sealants (Fayle 2003). Bonding of resin sealants to atypical etch patterns may result in reduction in longevity. Single-step adhesive systems have some evidence in greater retention at 70.2% at 4 years whilst three-step resin bonding systems had only 25.5% retention (Lygidakis, Dimou et al. 2009). Pre-treatment protocol with sodium hypochlorite found enhanced etching pattern using a single etch, but the evidence is limited to suggest this as routine for MIH sealants (Mathu-Muju and Wright 2006). Initial fissure sealing with GIC may be recommended in the presence of compromised moisture control, as in the case of a partially erupted MIH molar (Lygidakis, Wong et al. 2010).

3.4.3.2 Aesthetic concerns

For aesthetic concerns of anterior teeth, microabrasion, bleach and sealing of the hypomineralised area have been suggested with good preliminary success (Lygidakis, Wong et al. 2010). Microabrasion consists of an acidic pumice applied to remove the outermost 100µm of enamel over 1-2 appointments (Donly, O'Neill et al. 1992). Literature into the effectiveness of microabrasion and the aesthetic stability is limited, but improvements have been reported up to 1 month post appointment (Croll and Cavanaugh 1986). Creamy-yellow or whiteish-creamy MIH defects are less porous but variable in depth and may respond to microabrasion with 18% hydrochloric acid or 37.5% phosphoric acid and abrasive paste (Wray and Welbury 2001).

Bleaching can be applied to MIH to lighten the colour of the tooth surface. The released hydrogen peroxide anions, reactive oxygen molecules and free radicals is thought to be involved in improving the aesthetic appearance of enamel. Yellow or yellow-brown MIH defects are often full thickness and may respond to bleaching with carbamide peroxide (Fayle 2003). A combination with microabrasion has been investigated with good preliminary results. Resin infiltration may change the refractive index of hypomineralised lesions to create an acceptable appearance (Crombie, Manton et al. 2014). There are varied results for the effectiveness of restorative management to aesthetic concerns of MIH and further research is required. The low viscosity TEGMA resin may penetrate into the enamel with preliminary results suggesting to the level of the DEJ which may seal the affected resin from the oral environment and subsequently improve the mechanical properties.

3.4.3.3 Cavity design

Two approaches have been considered for cavity design in teeth with MIH; removal of all defective enamel or removal of only porous enamel until resistance to the bur or probe is felt (Lygidakis, Wong et al. 2010). Removal of all defective enamel provides sound enamel for bonding but excessive enamel is removed whilst removal of only porous enamel increases the risk of breakdown at the margins due to defective bonding. Amalgam restorations are not recommended (Kotsanos, Kaklamanos et al. 2005) and glass ionomer restorations have been recommended as an immediate but interim management strategy to reduce post-eruptive breakdown in non-ideal moisture control situations (Lygidakis, Wong et al. 2010).

3.4.3.4 Direct restorations

Restoration including adhesive and fissure sealants have been recommended prior to any post-eruptive break down occurs (Elhennawy and Schwendicke 2016). Poor annual retention rates of adhesive materials, both composite resin and glass ionomer cements, may be due to the porosity and disordered structure of the enamel in hypomineralised defects (Kotsanos, Kaklamanos et al. 2005). Amalgam was found to be unsuitable for restoration due to marginal leakage, poor insultation of the immature pulp and no support for the adjacent enamel (Fayle 2003). Suggestions of a pre-treatment application of sodium hypochlorite or use of a fifth generation bonding agent are yet to be validated (Mathu-Muju and Wright 2006, Lygidakis, Dimou et al. 2009). Self-etch adhesives remove the need to etch and rinse which may reduce pain during treatment for the hypersensitive tooth. Long-term retention of direct restorations appear limited but especially when the cavity design involves cusps or the marginal ridge of a molar (Elhennawy and Schwendicke 2016).

3.4.3.5 Preformed metal crowns

For teeth with moderate to severe MIH including cuspal involvement, preformed metal crowns have been a suggested treatment option (Fayle 2003). In a recent review of the literature (Elhennawy and Schwendicke 2016) two studies evaluated the effectiveness of preformed metal crowns as an alternative to direct restorations in MIH. The ease of application and the long term success was noted (Zagdwon, Fayle et al. 2003). The advantage of stainless steel

crowns is thermal protection to allow the pulp to mature, protection from PEB, maintenance of occlusion and vertical eruption. Success has been reported at 90% 4 years post treatment for first permanent molars with MIH (Kotsanos, Kaklamanos et al. 2005).

Assessment of composite, ceramic and case restorations is in the preliminary stages (Mejàre, Bergman et al. 2005) but with low quality evidence and limited long term studies in which each material has placed and followed over a reasonable time frame. Each technique requires cavity preparation which may include loss of otherwise healthy tooth structure. Minimally invasive restorations including cast onlay preparations found 98.2% success over 38.6 months (Gaardmand, Poulsen et al. 2013).

3.4.4 Extraction

Teeth with severe MIH may be considered for extraction to avoid the ongoing burden of dental procedures on a hopeless long-term prognosis tooth. Children with dental anxiety or their ability to cope with long-term treatment including complex long-term restorative management and orthodontic care may determine planning factors such as the use of sedation or general anaesthesia. Considering extraction for long-term management of MIH molars would necessitate prediction of the prognosis of the teeth and communication with an orthodontist. The condition of the second and third molars requires assessment and the patient's orthodontic alignment and occlusion need to be considered for an ideal treatment outcome. Multidisciplinary treatment planning and discussion with the parents is recommended prior to extraction of these teeth.

When conditions are favourable, space closure can be achieved when first permanent molars are extracted. Subsequent space closure occurred spontaneously more often in the maxilla than the mandible in children under 8 years (Eichenberger, Erb et al. 2015). Spontaneous space closure is unlikely to occur as favourably in older patients and orthodontic treatment may be needed to close unacceptable spaces (Elhennawy and Schwendicke 2016). Lower first molar extraction can result in tilting of the second molar due to the thinner lingual plate and subsequent scissor bite or unwanted tooth wear may occur. Ideal timing for lower extractions has been suggested at age 9-10 years of age when the bifurcation dentine of the second molar is mineralising with the beginning of root formation (Cobourne, Williams et al. 2009). Extractions of only one molar may result in a midline shift and balancing extractions should be considered (Çağlaroğlu, Kilic et al. 2008).

4. Conclusion

Early diagnosis of MIH allows preventative or early restorative management to prevent the need for more invasive and permanent treatment modalities. Assessment of deciduous teeth for developmental defects of enamel may be an indication of a disturbance which has the potential for carious changes in the primary dentition or MIH of the permanent dentition. A short recall would be recommended for these patients to establish preventative measures very early during eruption. Hypersensitivity requires immediate management to reduce the risk of caries or dental anxiety. More severe molar defects may be managed with direct restorations or preformed restorations or extractions for the most severe cases in conjunction with an orthodontic management plan. The evidence for these clinical suggestions come mainly from observational studies and further research requires more stringent internal and external validity before management protocols are clear.

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Child Protection: It's All of Our Business

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"All children have the right to be protected from all forms of negligent treatment and enjoy the highest attainable standards of health"

– United Nations Convention on the Rights of the Child[1]

Introduction

Paediatric dentists are highly trained specialists, proficient at performing comprehensive dental evaluation, treatment and education of children and their families. As dental examination and treatment involves thorough investigation of medical, dental, social and behavioural histories of the child and family unit, often carried out over a number of regular visits, paediatric dentists and their staff are in an optimal position to recognise risk factors, signs and symptoms of child maltreatment. They have a responsibility towards promoting child safety and welfare, recording and reporting adverse events, educating colleagues and supporting families to reduce the incidence and consequences of child maltreatment, which can include medical and dental mismanagement. This review will define the problem of child maltreatment and how it relates specifically to the paediatric dental profession and will outline a protocol for management if child abuse, neglect or maltreatment should be suspected in the dental setting.

So, what's the problem?

The Australian Institute of Family Studies defines child maltreatment as "any non-accidental behaviour by parents, caregivers, other adults or older adolescents that is outside the norms of conduct and entails a substantial risk of causing physical or emotional harm to a child or young person"[2]. Five subtypes are recognised: physical, emotional and sexual abuse, neglect and exposure to family violence. These subtypes are defined and described in Table 1. Other forms of child maltreatment identified in the literature that currently stand outside the five main subtypes include fetal abuse behaviours of pregnant mothers, peer and/or sibling abuse including bullying, institutional abuse, child trafficking and exposure to community violence[3].

Child maltreatment is a significant global problem; the World Health Organization[4] estimates one in four adults have a history of physical abuse as a child and one fifth of women have been sexually abused. Unfortunately, classifying and recording types of child maltreatment is not straightforward, as it is known to be under-reported, difficult to classify and studies tend to lack methodologic rigour and validated measurement tools[5]. Additionally, it can be difficult to delineate abuse from culturally and developmentally-determined norms in different populations and determine which parental behaviours are appropriate in their specific context.

In Australia, the best available indicator and only routine data collection regarding child maltreatment is child protection statistics, which only calculates the number of children who have had encounters with child protection services, which is likely to underestimate the problem[6]. The most recent data from the Australian Institute of Health and Welfare[7] identifies that the number of children subject to child protection services has risen over the past 5 years, reaching one in 32 Australian children. A recent systematic review[8] identified neglect as the most regularly reported type of maltreatment in the USA and UK, whereas in Australia,[7] emotional abuse was found to be the most common. Child abuse and neglect can have a number of significant consequences on physical, biological, emotional, social and cognitive development of victims, with increased risk of depressive and anxiety disorders and intentional self-harm[9]. The World Health Organization[4] estimates 41,000 child homicides occur annually, however, the actual number of child deaths associated with child abuse or neglect is likely to be more than three times this figure, as many child maltreatment deaths are wrongly classified as outcomes of other causes.

How is this relevant to the paediatric dentist?

"67% of dentists with an interest in paediatric dentistry had suspected abuse or neglect of a child patient, but only 29% had ever made a child protection referral"
– Harris et al.[16]

Table 1. *Child maltreatment definitions and example behaviours by subtype*

	Physical abuse	Emotional abuse	Sexual abuse	Neglect	Exposure to family violence
Definitions	Non-accidental employment of physical energy that culminates in actual or potential har[2]	Inappropriate ongoing verbal or non-verbal patterns that fail to provide appropriate nurturing and emotional sustenance[2]	An activity that goes against what is socially acceptable in society, involving a child who does not fully understand its nature or implications and is unable to give informed consent[10]	Caregiver fails to facilitate essential physical and emotional care, when they have the resources and ability to do so[11-14]	Child witnessing, hearing or otherwise being exposed to the event or after effects of physical, sexual or emotional abuse of family members[15]

Considering the propensity for missed opportunities for interception of child maltreatment, in particular due to differing reporting systems and regulations in the various states and territories of Australia, the Council of Australian Governments in 2009 developed the National Framework for Protecting Australia's Children[17]. This framework promotes a preventive model, whereby everyone in the community shares responsibility for identification of potential child maltreatment and appropriate reporting, referral and support of families. The aim to is avoid a crisis-driven system that is only operative after significant damage has been sustained in children and families. The Royal Australasian College of Physicians[18] also recently launched a child protection policy incorporating the notion of utilising the whole community for multiple preventive interception opportunities.

Paediatric dentists have the privilege and responsibility of spending regular time, and forming close relationships, with their child patients and their families. These interactions allow for regular examinations of children as well as opportunity to observe family dynamics. Along with this comes the moral and ethical obligation that the dental professional is able to recognise and react accordingly to signs of child maltreatment or, more rarely, open disclosure by the child. Additionally, in Australia, under the National Law, all dental practitioners are subject to report conduct of other practitioners that places the public at risk of harm due to intoxication, sexual misconduct, or deviation from acceptable professional standards[19]. The paediatric dentist may be required to contribute their skills to diagnosis, assessment and planning when concerns have been raised as to the child's welfare or previous dental treatment and participate in rehabilitation of oral neglect or injury. Paediatric dental specialists would also be expected to play a role in mentoring and education of dental and non-dental professions with regular contact with children to ensure that oral signs of maltreatment are not missed or incorrectly diagnosed and integrate oral health into multidisciplinary assessment and planning of maltreated children.

Victims of child abuse are known to have poorer oral health and associated physical and psychological consequences, often requiring the skills of the paediatric dentist in their management[20-25]. The same characteristics that cause children to seek specialist dental care, including complex medical, behavioural or developmental conditions, also place children at higher risk of maltreatment and neglect, providing further opportunity for paediatric dentists to lead safeguarding of children[26]. Finally, it is in the paediatric dentist's best interest that children thrive in optimal physical and emotional family environments, as this has been associated with reduced risk of dental disease and associated treatment burden and cost, improved compliance with oral health education and reduced behavioural difficulties in the dental environment[27-30].

Dental neglect

Both dental health and care are essential components for children's overall health and wellbeing. Failure of caregivers to pursue appropriate dental advice, prevention or treatment for their child can be considered dental neglect, which has been acknowledged as a problem common to children of all ages[31-35]. The American Academy of Paediatric Dentistry and The British Society of Paediatric Dentistry definitions of dental neglect are displayed in Table 2. There are a number of acknowledged barriers to the identification and reporting of dental neglect, including the high prevalence of dental caries, complex aetiology and lack of standardised guidelines to distinguish purposeful neglect from socio-economically, geographically and culturally determined behaviours[8,27,29,30]. The impact of oral disease on the child, risks and benefits of treatment, family's ability to understand advice and access care and the child's capability for accepting dental treatment all need to be carefully assessed in order to diagnose dental neglect[36,37]. Early recognition of dental neglect can be valuable in prompting psychosocial evaluation of the family and interventions to educate, support and assist in determining need for further investigation into broader neglect[38]. The numerous long-term consequences of untreated caries on children's health and wellbeing are well known; however, it is reassuring that, after appropriate dental care is provided, children can exhibit improved growth and quality of life[23, 39-43].

Issues of consent are often raised in situations of potential dental neglect or when children are subject to child protection or custody orders. For children younger than 18 years, the ability of the child to consent varies by jurisdiction in Australia and dental practitioners should refer to the Australian Dental Association[44] guideline that outlines consent procedures. Where there is uncertainty, practitioners should contact Legal Services or the Child Protection Litigation Office for advice[45]. Failure to obtain appropriate consent from both the caregiver and the child can lead to allegations of assault, negligence or professional misconduct. Consent must also be gained prior to conducting examinations to document suspected abuse[46].

How can I recognise non-accidental injuries and neglect in the dental environment?

Indications of physical and sexual abuse, as well as ailments associated with emotional abuse and neglect, are commonly observed in the oral cavity, with the head and neck being involved in up to three quarters of physically abused children and the lips being the most common orally involved tissue[48-53]. Intra-oral injuries are less common, however, it is likely that these are significantly underestimated as they are more easily hidden and not always

Table 2. *Definitions of dental neglect*

American Academy of Pediatric Dentistry[47]	"Wilful failure of parent or guardian to seek and follow through with treatment necessary to ensure a level of oral health essential for adequate function and freedom from pain and infection"
Harris[37] (British Society of Paediatric Dentistry)	"The persistent failure to meet a child's basic oral health needs, likely to result in the serious impairment of a child's oral or general health or development"

Table 3: *Signs and symptoms of child maltreatment that might become apparent during dental visits*[8,18,31,60-65]

Signs and symptoms	
Medical history	• Multiple unexplained hospital visits for injuries or medical treatment or use of multiple different doctors/hospitals • Caregiver ignorance of or inconsistencies regarding important medical conditions, treatment or events • Diagnosis and/or treatment of depression, eating disorders or other psychological disorders • Children supplied with inappropriate medications or drugs by caregiver • Pregnancy
Dental history	• Multiple unexplained incidences of dental trauma or treatment • Irregular/delayed examinations and/or treatment in the presence of obvious dental disease • Reluctance to give information, ignorance, denial or conflicting versions of past injuries or treatment • Trauma history is inconsistent with child's physical and/or developmental characteristics
Social history	• Lack of attendance at educational facilities • Children not meeting appropriate developmental milestones • Lack of age-appropriate interests and social interaction • Spends long periods of time unsupervised that are not age or developmentally appropriate • Inappropriate sexual behaviour or knowledge • Evidence of substance abuse
Oral health behaviours	• Caregiver ignorance or refusal to assist with or provide tools for age-appropriate basic oral hygiene • Caregiver ignorance or deliberate failure to provide a nutritious diet
Extra-oral examination	• Single or multiple extra-oral injuries that are: • Unexplained or with an inconsistent history • Delayed in presentation • Untreated even though significant • Abnormal in location (ears, side of face, neck top of shoulders, inner aspects of arms, back and side of trunk, cheeks, forearms, chest and abdomen, groin, inner thighs, soles of feet) • Bilateral • Display particular patterns or show the shape of any object (eg: bite marks, cigarette burns) • Not consistent with child's age (eg: falls in children who are not yet walking) • Of varying stages of healing • Obviously self-inflicted • Obvious deficiencies in clothing, hygiene or wearing of inappropriate clothing (eg: long sleeves and pants in hot weather) or reluctance to remove clothing • Evidence of malnutrition, failure to thrive and abnormal growth • Difficulties in walking, sitting
Intra-oral examination	• Atypical intra-oral injuries not consistent with history • Lacerations or oral frena (often occurs with other findings of serious physical abuse), palatal petechiae, mucosal tears, bizarre outlines or semblance to traumatic object insertion • Evidence of repeated oral injury without treatment • Evidence of sexually transmitted oral or peri-oral infections (eg: gonorrhoea, warts, syphilis) • Evidence of malnutrition and/or poor diet (mucosal ulceration, burning, extensive dental caries, periodontal problems) • Evidence of significant untreated oral disease, trauma, pain and/or infections
Radiographic examination	• Evidence of unexplained healed or unhealed tooth, root or bony fractures • Evidence of insertion of penetrating objects
Treatment planning and dental treatment	• Caregiver seems uninterested or unconcerned in the presence of significant dental injury, disease or treatment required • Caregiver declining or failing to follow through recommended treatment without clear and warranted justification • Caregiver threatening the child • Caregiver exhibiting unrealistic expectations of the treatment or the child's response or behaviour towards it • Excessive unexplained daytime sleepiness, inability to concentrate, headaches, anxiety, attention-seeking or aggressive behaviour during treatment • Child showing abnormal responses to pain • Excessive friendliness to strangers, wariness, flinching or fearful to caregivers, other people or loud noise, not wanting to go home • Abnormal attachment with caregivers – trying too hard to please or failure to connect • Extremes of behaviour – aggressive to passive, defiant to over-eager to please • Frequently late or miss appointments despite reminders, advice and explanation of the need for treatment

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fully explored[54]. This is evidenced by the fact that when assessment of physically abused children involves forensic dentists, much higher prevalence of intra-oral injuries is recorded, highlighting the essentiality of integrating dentists into medical assessment pathways for abused children[55, 56].

All members of the dental team should routinely be vigilant towards signs and symptoms that might indicate child maltreatment[57]. Careful observation of the child and family by reception staff as they enter the dental office and interact in the waiting room might identify at-risk and atypical behaviours. Apprehension, withdrawal, anxiety and agitation are, unfortunately, commonly exhibited behaviours of fearful children in the dental office, however, if accompanied by obvious aggressive or denigrating behaviour of caregivers or appears out of context with the situation, then further investigation is warranted. Occasionally, dental assisting staff might observe behaviours that are deliberately kept hidden from the dentist.

Accidental trauma to the face and oral cavity are common reasons for presentation to a paediatric dental office; worldwide epidemiological studies highlight that approximately one third of preschool children will sustain injuries to their primary dentition with a quarter of school children suffering the same to their permanent dentition[58]. Injuries need to be analysed in context of the patient's medical, dental and social history, specific history and circumstances of the event and the child's physical and developmental characteristics. A case-control study reported over one quarter of infants presenting with severe signs of physical abuse had evidence of one or more previous sentinel injuries, the most common being bruises and intra-oral injuries, highlighting the importance of detecting minor abusive injuries in the dental setting and providing the appropriate interventions to avoid further devastating harm to the child[59,60]. Table 3 outlines signs, symptoms and risk factors of child abuse and neglect that the dental team might identify during history taking, examination and discussion with the child and family. All observations and discussions should be meticulously documented and witnessed by other staff.

What should I do if I suspect a child or adolescent is a victim of maltreatment?

Dental practitioners are likely to feel uncertain about identifying and reporting suspected child abuse, as often cases go undetected due to the hidden nature of the crime, difficultly in disclosure by the child and lack of clear thresholds required to substantiate the claims[61]. Research into reporting of child abuse and dental neglect identified a lack of clear definitions, protocols, knowledge and confidence of reporting pathways, fear of harming professional and personal relationships and fear of physical, financial or legal retribution against the child or dental staff[8,31]. Considering the potentially devastating implications for children and families of unidentified child maltreatment, practical child protection and safeguarding protocols, education and training should be implemented in all organisations that care for children, including both public and private dental practices.

Child protection is certainly not yet an evidence-based practice, so it is essential that individual organisations develop clearly defined protocols, training and performance review to ensure staff competency in managing child maltreatment. In the United Kingdom, a dental-specific government funded child protection educational resource demonstrated significant improvements in practitioner confidence, referrals and changes in practice[67,68]. Although the Australian literature is somewhat lacking in this regard, guidance can be taken from policy documents developed by the American Academy of Pediatric Dentistry[57], National Institute for Health and Care Excellence[64] and the British Society of Paediatric Dentistry[37]. Child protection protocols should promote the early recognition of risk factors, signs and symptoms of child abuse and neglect and provide unambiguous pathways for reporting and referral, specific to each jurisdiction. The following protocol, described briefly below and displayed in Flowchart 1, is specific to dental settings in Victoria; for comparison, Table 4 briefly outlines pathways applicable to other states and territories of Australia. Diagram 1 illustrates a body and mouth map that could be utilised to record location, size and shape of injuries[69].

When deciding what steps to take when confronted with potential child maltreatment, the practitioner should always consult wider organisational policy and senior medical, dental, social work and psychology colleagues. Paediatric dental specialists should also make their medical colleagues aware of the vital input they may have in the examination, treatment and follow-up of children affected by abuse or neglect and ensure that dental clinical notes are integrated into the child's electronic medical record. The priorities in managing child abuse are firstly to diagnose, treat and document the child's injuries, interpret patterns of injury or behaviour preceding the suspicion of child abuse, to notify and involve the relevant child protection and paediatric medical services and prepare detailed written documentation[46]. The type of suspected abuse will dictate the urgency of any referrals; immediate referral is usually required for suspected sexual abuse, as accuracy of diagnosis and maintenance of a chain of evidence is increased if evidence is collected within 24-72 hours by specialist personnel[57,70]. Physical abuse may also require urgent referral to an emergency hospital setting, especially if the injuries are outside the scope of dental practice. When the dental team is confronted with suspect child maltreatment, it is imperative that both families and staff feel safe to communicate openly about their situations and opinions, which should be documented and witnessed carefully and, where available, involve senior medical and psychology providers and ethics committees[46, 71].

Curiously, in Victoria, as in Queensland and Western Australia, dentists are not specifically listed as mandatory reporters of suspected child maltreatment, although doctors, nurses, police officers and school teachers are[72]. However, dental professionals have an ethical and moral obligation to ensure child protection and the Victorian Department of Health and Human Services recommends that anyone should contact the Child Protection service if they have reasonable grounds for believing a child less than 18 years has suffered or is suffering significant harm[72, 73]. Additionally, the new Crimes Amendment (Protection of Children) Act 2014 mandates that it is a criminal offence for any adult to delay or fail to disclose any reasonable suspicion that a child under 16 years of age in Victoria is experiencing sexual abuse. It should also be emphasised that, in all jurisdictions, it is not the responsibility of the reporter to further investigate after reporting, reporting requirements are prioritised over professional codes of practice and any person who discloses information in good faith is treated with confidentiality and protected from any civil, criminal or administrative proceedings. The Australian Institute of Family Studies[74] provides an excellent resource regarding collection of information, mandatory reporting and actions to take if a child discloses maltreatment. After reporting to child protection services, the dental practitioner has a responsibility to participate in the ongoing care, documentation and follow-up of the child through legal proceedings and beyond.

Table 4: *Reporting requirements and child protection contact details in Australia (adapted from Australian Institute of Family Studies[74])*

State/Territory	Reporting requirements	Child protection reporting authority and contact details
Victoria	• Dentists are not specifically listed as mandatory reporters for children 0-17 years,however registered medical practitioners, nurses, midwives, teachers, early childhood teachers and police officers are • All adults are mandated to report sexual abuse for children under 16 years	Victorian Child Protection Service **Ph: 13 12 78**
ACT	• Dentists are mandated reporters for children 0-17 years • Physical and sexual abuse must be reported	Child and Youth Protection Services **Ph: 1300 556 728**
NSW	• Dentists are mandated reporters for children 0-15 years • All subtypes of abuse and neglect must be reported	Department of Family and Community Services Child Protection Helpline **Ph: 13 21 11**
NT	• Every person is required to report for children 0-17 years • All subtypes of abuse and neglect must be reported	Territory Families Child Protection Hotline **Ph: 1800 700 250**
QLD	• Dentists are not specifically listed as mandatory reporters for children 0-17 years, however doctors, teachers, police officers and early childhood educators are • Physical and sexual abuse must be reported	Department of Communities, Child Safety and Disability Services **Ph: 1800 177 135**
SA	• Dentists are mandated reporters for children 0-17 years • Physical, sexual, emotional abuse and neglect muse be reported	Department of Child Protection Child Abuse Report Line **Ph: 13 14 78**
TAS	• All adults in Tasmania have a responsibility to report for children 0-17 years • All subtypes of abuse and neglect must be reported	Children and Youth Services **Ph: 1300 737 639**
WA	• Dentists are not specifically listed as mandatory reporters for children 0-17 years, however doctors, nurses, midwives, teachers, boarding supervisors and police offices are mandated to report sexual abuse • Additionally, registrars, family counsellors/dispute resolution practitioners or legal practitioners are mandated to report all subtypes of abuse and neglect	Department of Communities, Child Protection and Family Support **Ph: 1800 199 008**
ALL STATES	**Dental Board of Australia[73] Code of Conduct:** "Good practice involves ensuring that, when communicating with a child or young person, practitioners remain alert to children and young people who may be at risk and notify appropriate child protection authorities as required by law. This may include where a parent or guardian is refusing treatment for their child or young person and this decision may not be in the best interests of the child or young person"	

Conclusion

Child maltreatment is reaching endemic proportions in our society and burdens children and their families with lifelong consequences. Both child maltreatment and oral diseases share similar risk factors including educational, social and financial disadvantage, contribute to reduced quality of life, physical and mental health. Paediatric dentists arguably underestimate their essential contributions to prevention, identification, management and follow-up of child maltreatment and associated oral conditions. These dental specialists must hold practical knowledge of the local services, support and referral pathways available for children and families and liase with other children's professionals to develop targeted, multidisciplinary protocols for prevention, intervention and optimisation of oral and general health in this vulnerable population.

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Flowchart 1. *Protocol for assessing and reporting suspected child maltreatment in dental settings in Victoria, Australia*

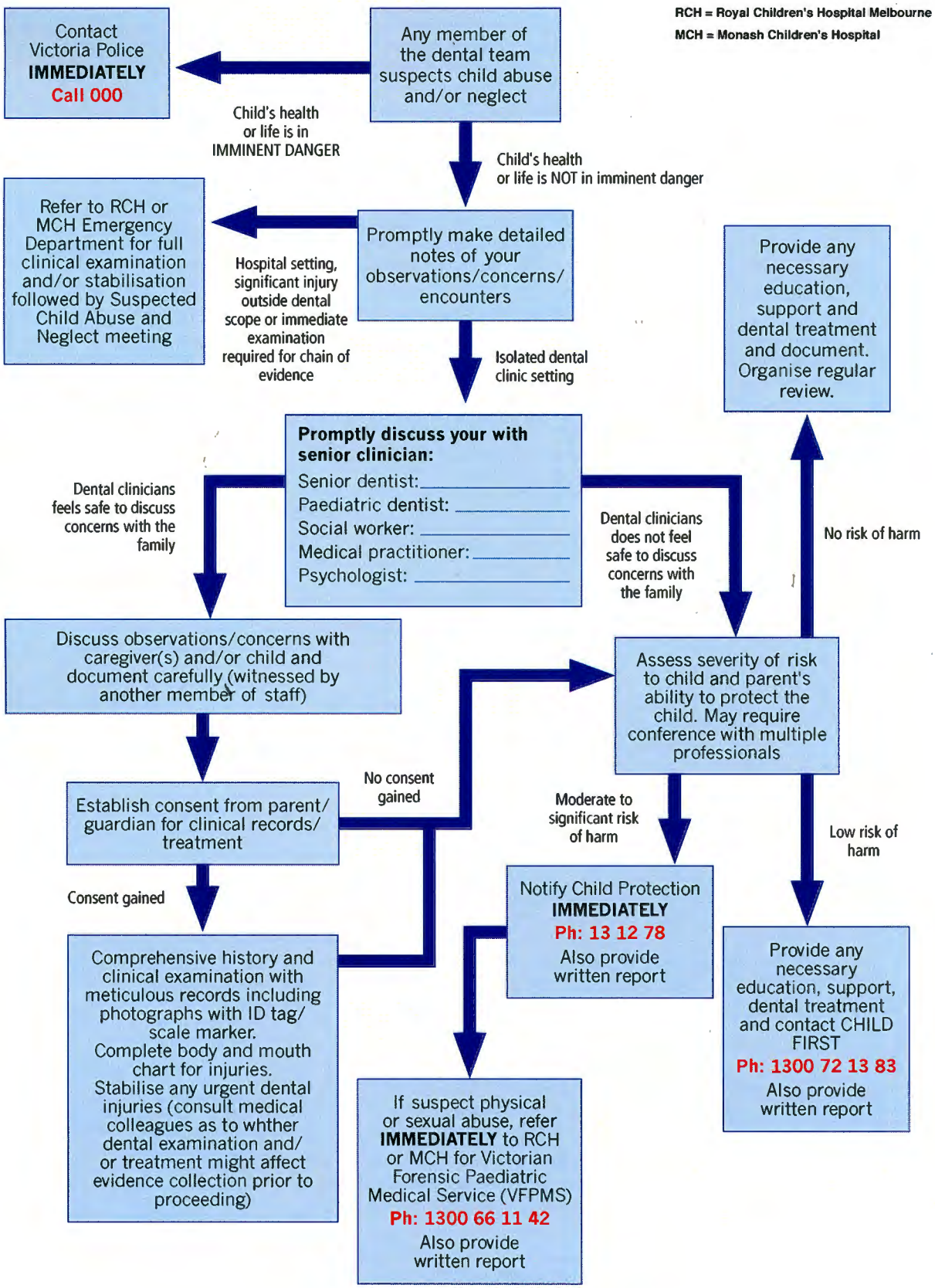
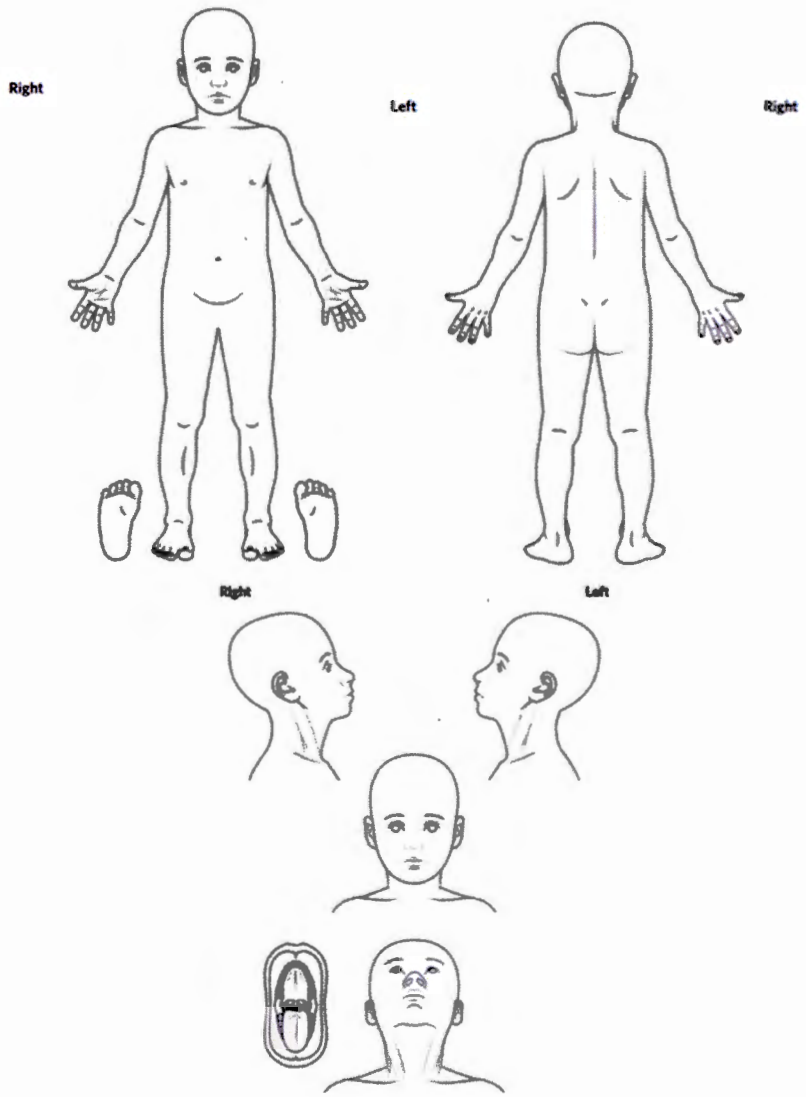


Diagram 1. *Body and mouth map for recording injuries and evidence of maltreatment (adapted from Victorian Forensic Paediatric Medical Service[69])*



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ANZSPD RK Hall Lecture Series
Perth, WA

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Adelaide Convention Centre
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annual.aapd.org

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27th IAPD Cancun Congress
Cancun, Mexico
www.iapd2019.org

7-12 June 2021
28th IAPD Maastricht Congress
Maastricht, The Netherlands
www.iapd2021.org

Apology

The previous issue of Synopses (August 2018, Issue 63) contained an article commencing on page 8, entitled "The oral biofilm in paediatric patients". The author, whose name was omitted from the article, was Dr Lydia Ng, second year postgraduate student in Adelaide.